Equine Colic

Interactive CD & Supplemental Text

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Equine Colic

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Sisson and Grossman's *The Anatomy of the Domestic Animals*, Robert Getty.

The Anatomy of Domestic Animals: The Viscera, Richard Nickel, August Schummer, Eugen Seiferle.

Illustrated Veterinary Anatomical Nomenclature, Oskar Schaller.

Equine Colic

Supplemental Text

Supplemental text written by Dr. James Moore
Booklet designed and set by Melissa Bugbee Buchanan

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The Language of Anatomy

For you, the user, to get the most out of the three-dimensional anatomical images that support this program, the accompanying descriptions must be as unambiguous as possible. To this end, we have used anatomical names from recent equine anatomy textbooks and common names where appropriate. In addition, we have included on the CD-Rom an introductory movie illustrating eight important directional and positional terms that will be used throughout the rest of this program.

The first two terms, **medial** and **lateral**, are introduced by passing sagittal planes through the horse from head to tail. One of these planes, called the median plane, passes along the midline, dividing the body into right and left halves; other sagittal planes would be positioned parallel to the median plane. Structures that are closer to the median plane are termed medial (Figure 1), and structures that are farther from the median plane are termed lateral (Figure 2).

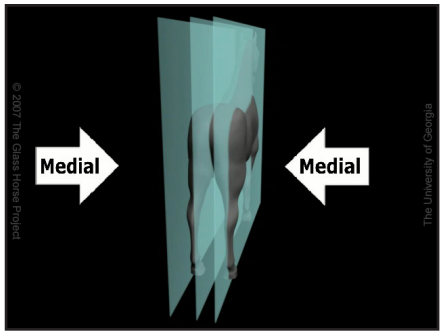

Figure 1. Towards the median plane is medial.

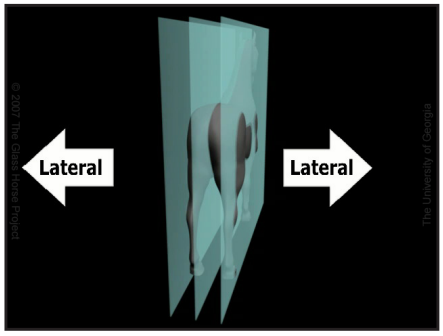

Figure 2. Away from the median plane is lateral.

The second two terms, **dorsal** and **ventral**, are introduced by passing planes, called dorsal planes, through the body parallel to the ground surface. Using one of these planes as a point of reference, structures above the plane are termed dorsal (Figure 3), and structures below the plane are termed ventral (Figure 4).

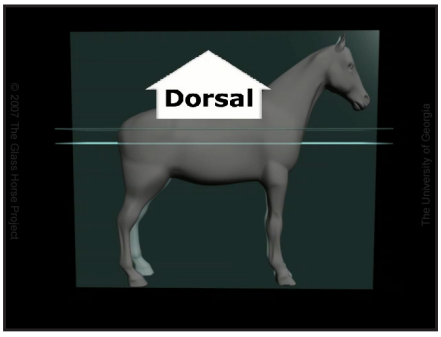

Figure 3. Above a dorsal plane is dorsal.

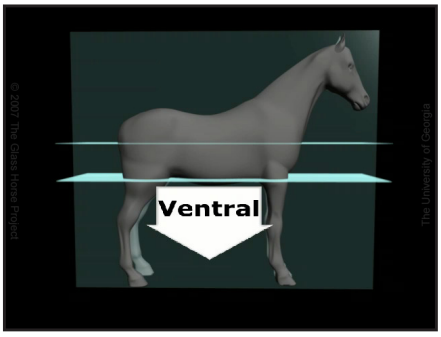

Figure 4. Below a dorsal plane is ventral.

The third set of terms, **cranial** and **caudal**, are introduced by passing a transverse plane through the body perpendicular to both the median and dorsal planes. Structures towards the horse's head are referred to as cranial (Figure 5), while those towards the horse's rear are referred to as caudal (Figure 6). These two terms are also used to describe the relative positions of points along portions of the gastrointestinal tract. For instance, the initial portion of the duodenum is called the cranial duodenum, and the latter part of the duodenum is called the caudal duodenum.

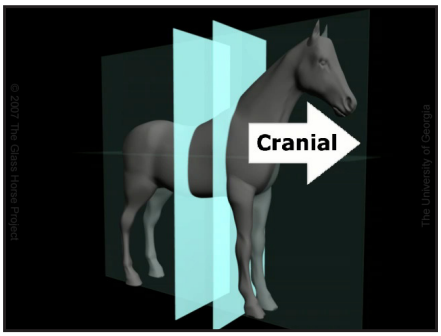

Figure 5. Towards the horse's head is cranial.

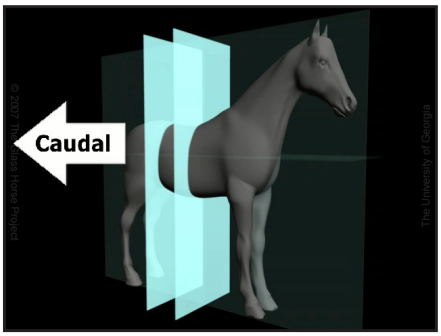

Figure 6. Towards the horse's tail is caudal.

The last two terms, **orad** and **aborad**, refer to the flow of ingesta through the gastrointestinal tract; orad means moving towards the mouth, and aborad means moving away from it. Don't be shocked to find that many clinicians use the term *proximal* instead of orad and *distal* instead of aborad. Strictly speaking, proximal and distal are used to describe the relative positions of structures on the extremities.

The Horse's Gastrointestinal Tract and Abdomen

Introduction

Some people develop a good mental image of the layout of the horse's gastrointestinal tract by visualizing the tract put together piece by piece. A logical method for doing this is to follow the same path taken by feed material, otherwise known as ingesta. This can be achieved best by starting with an empty abdomen bounded by a transparent rib cage and vertebral column.

The **stomach** is positioned primarily on the left side of the cranial aspect of the abdomen (Figure 7). From the stomach, the ingesta moves into the **duodenum**, which is attached to the dorsal body wall on the right side of the abdomen by a short mesentery (Figure 8). At about the level of the horse's flank, the duodenum changes direction, turning to the left. Just after it crosses the midline, the mesentery supporting the duodenum lengthens considerably, demarcating the transition to the next part of the small intestine, the **jejunum** (Figure 9). In the normal adult horse, the jejunum is about 20 meters long. However, to simplify the situation, this entire length has not been incorporated into the model. The last part of the small intestine is the thick, muscular **ileum** (Figure 10).

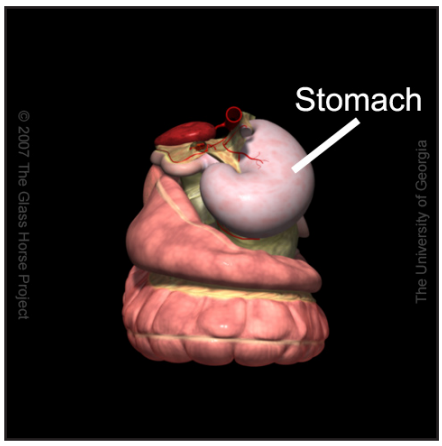

Figure 7. The stomach is positioned dorsally in the abdomen.

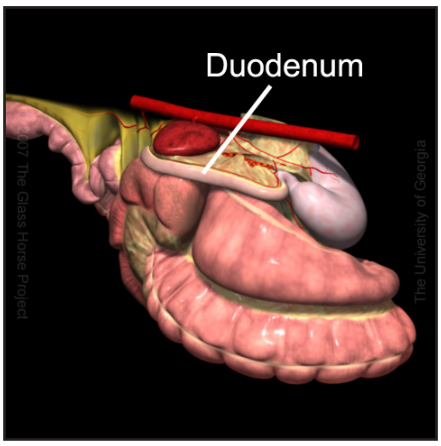

Figure 8. The duodenum is positioned dorsally on the right side of the abdomen.

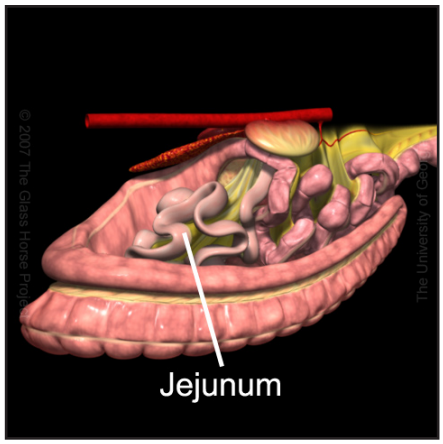

Figure 9. The jejunum has a long mesentery.

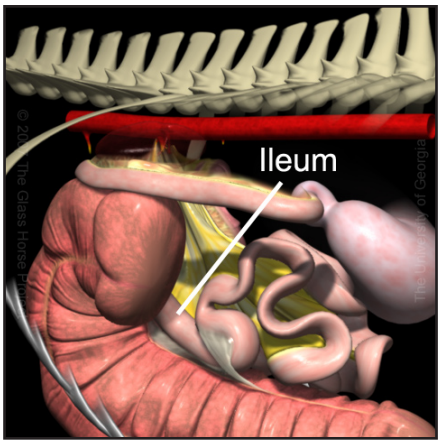

Figure 10. The terminal part of the small intestine is the ileum.

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The ingesta leaves the ileum and passes through the ileocecal orifice into the **cecum**. This junction between the small intestine and cecum functions as a one-way valve to control the flow of ingesta. The ingesta then enters the cecum, which is a large, comma-shaped organ that is positioned primarily on the horse's right side, extending from the right flank region to the sternum (Figure 11). The ingesta is mixed within the cecum by waves of muscular activity that originate near the ileocecal junction, move through the body to the apex, and then return towards the cecal base. Ingesta leaves the cecum through the cecocolic orifice, and enters the large colon. It enters the **right ventral colon** and then it moves around to the **left ventral colon**, up into the **left dorsal colon** (Figure 12), and finally back around into the **right dorsal colon** (Figure 13). From there, the ingesta passes through the short **transverse colon** (Figure 14) and into the **small colon**, where fecal balls are made (Figure 14). Finally, undigested waste material leaves the body through the **rectum** (Figure 14).

The other organs in the abdominal cavity include the paired left and right kidneys, the spleen, the liver, and the pancreas, the blood vessels that supply them, and the special folds of peritoneum that connect these organs to each

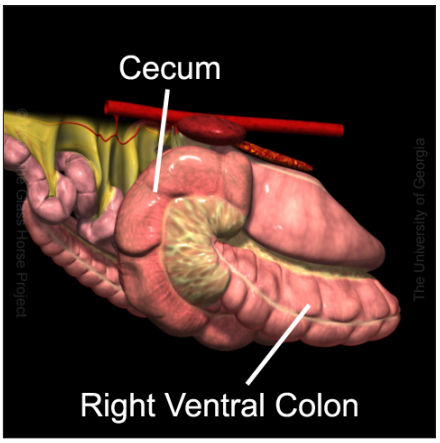

Figure 11. The cecum and right ventral colon are on the horse's right side.

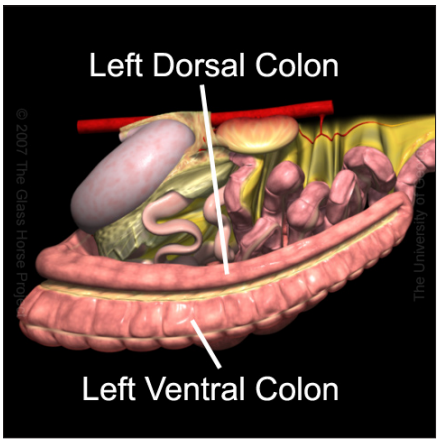

Figure 12. The left ventral and left dorsal colons are on the left side.

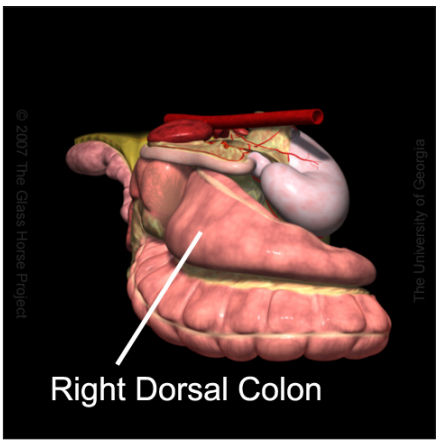

Figure 13. The right dorsal colon is positioned dorsally on the right side.

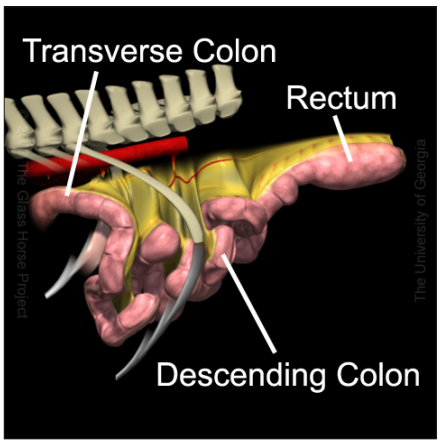

Figure 14. The transverse, small colon, and rectum viewed from the left side

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other and to the body wall. The **left kidney** (Figure 15) is positioned a bit further caudally than the **right kidney** (Figure 16) and hangs obliquely from the dorsal body wall. Near the left kidney is the **spleen**; these two organs are attached by the **renosplenic ligament**. The **liver** (Figure 17) is positioned in the dorsocranial aspect of the abdomen, between the stomach and the diaphragm, and the **pancreas** (Figure 18) is adjacent to the liver and the initial portion of the duodenum.

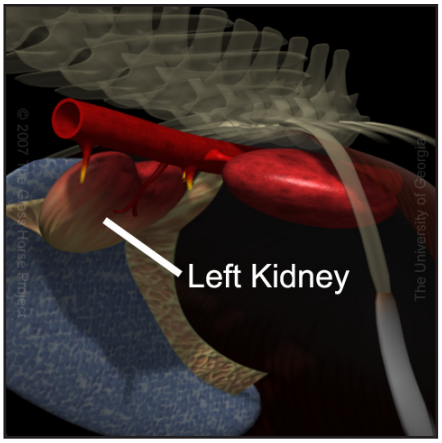

Figure 15. The left kidney is more caudal than the right kidney.

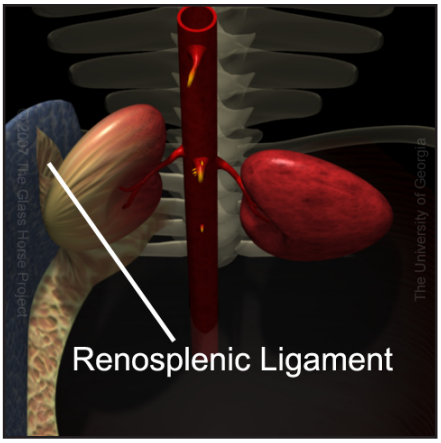

Figure 16. The renosplenic ligament connects the spleen and left kidney.

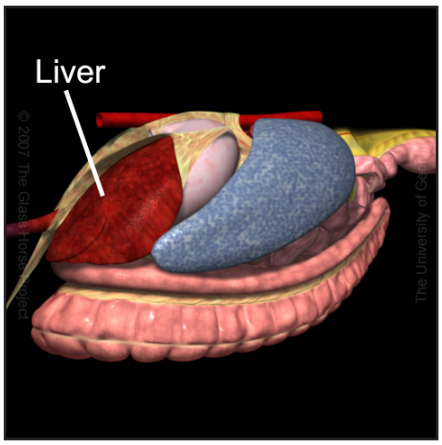

Figure 17. The liver is positioned between the stomach and diaphragm.

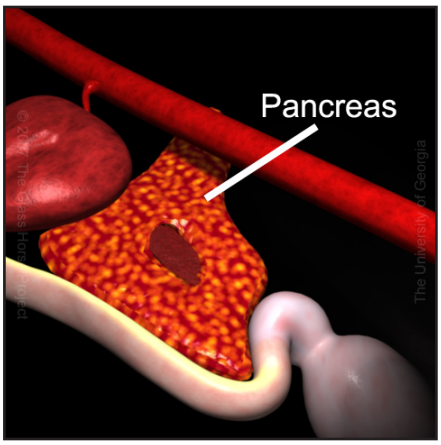

Figure 18. The pancreas is adjacent to the liver and duodenum.

Stomach

The horse's stomach is relatively small, with a capacity of approximately 10 to 15 liters, and is situated dorsocranially in the left side of the abdomen within the confines of the ribcage. Due to the shape of the stomach, the junction between the esophagus and the stomach, called the **cardia**, is near the pylorus, which connects the stomach to the small intestine. The oblique nature of the cardia renders it virtually impossible for the horse to vomit and thus makes passage of a nasogastric tube a critical component of the diagnostic work up of horses with colic.

The stomach is comprised of different regions and curvatures (Figure 19). The **fundus** is the blind-ended, dorsal sac of the stomach. The **body** is the largest portion of the stomach, extending from the fundus on the left to the pylorus on the right. The **pylorus**, which is the muscular junction between the stomach and the duodenum, is characterized externally by a distinct constriction in size.

The stomach has two curvatures (Figure 20). The **lesser curvature** of the stomach is the short distance from the cardia to the pylorus. The greater curvature of the stomach extends dorsally from the cardia to the left side, continues ventrally, and then returns back to the right side to the pylorus. The **greater curvature** of the stomach is attached to the hilus of the spleen by the **gastrosplenic ligament**, and the **gastrophrenic ligament** attaches the greater curvature of the stomach to the crura of the diaphragm.

The stomach also has two omenta: the greater omentum and the lesser omentum. The **greater omentum** (Figure 21) is the thin fold of peritoneum that loosely connects the ventral aspect of the greater curvature of the stomach and the initial curve of the duodenum with the right dorsal colon, transverse colon, and proximal portion of the descending colon. The greater omentum, together with the gastrophrenic, gastrosplenic, phrenicosplenic, and renosplenic ligaments, forms a large loose sac. The **lesser omentum** (Figure 22) is formed by the hepatogastric and hepatoduodenal ligaments, which connect the stomach and duodenum with the liver. As

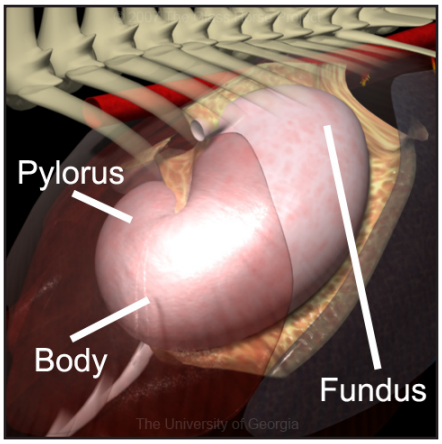

Figure 19. The fundus, body, and pylorus of the stomach

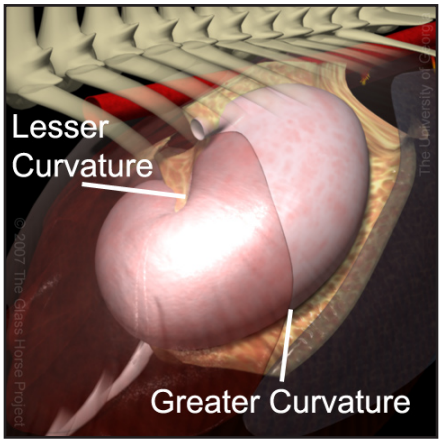

Figure 20. The lesser and greater curvatures of the stomach

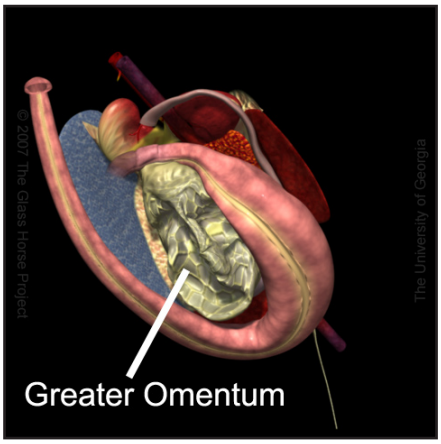

Figure 21. Attachment of the greater omentum to the colons

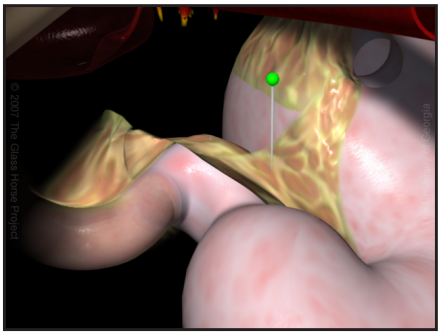

Figure 22. The lesser omentum is attached to the stomach.

such, the lesser omentum comprises a portion of the wall of the vestibule of the **omental bursa**. The caudal recess of the omental bursa is a relatively large cavity caudal to the stomach that is enclosed by the greater omentum.

Small Intestine

The small intestine, which in the adult horse totals approximately 20 meters in length, is comprised of the duodenum, jejunum, and ileum. The duodenum is the first part of the small intestine and is situated on the right side of the abdomen. The **duodenum** is comprised of two flexures, a descending portion and an ascending portion (Figure 23). The cranial part of the duodenum, which is adjacent to the liver, has an initial sharp curve and a dilated ampulla. The second curve in the duodenum, which is called the **cranial flexure**, encompasses the pancreas. This part of the duodenum is attached to the liver by the **hepatoduodenal ligament**, which contains the pancreatic and bile ducts (Figure 24). The **descending duodenum** passes dorsocaudally and is attached to the body wall by a short mesentery, called the **mesoduodenum**. The bile duct, which is also called the common hepatic duct, passes between two layers of mesoduodenum to enter the lumen of the duodenum approximately 15 cm from the pylorus. At its caudal flexure, the duodenum passes around the base of the cecum and crosses the midline caudal to the root of the mesentery. The **ascending duodenum** then extends for a short distance in a cranial direction (Figure 25). At this point, the duodenum is connected to the transverse colon and initial portion of the descending colon by a portion of the mesoduodenum called the **duodenocolic fold**. This duodenocolic fold is often used during abdominal surgery as a landmark for identifying the duodenum.

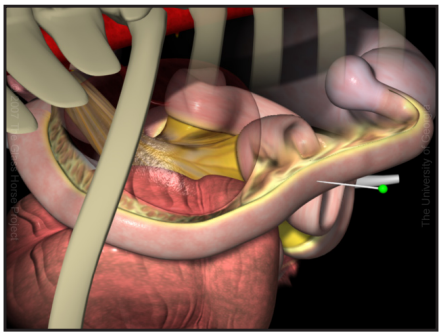

Figure 23. The descending duodenum on the horse's right side

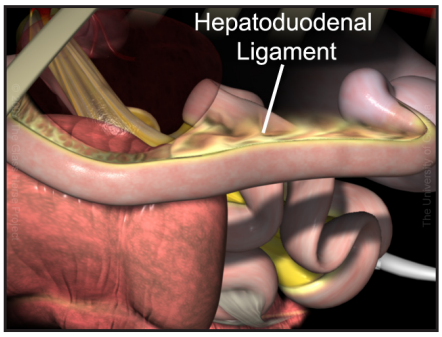

Figure 24. The hepatoduodenal ligament connects the duodenum and liver.

The second part of the small intestine is the **jejunum**, which lies in coils in the middle of the abdomen (Figure 26). The length of the mesentery increases markedly in the jejunum, creating a situation in which loops of the small intestine may become incarcerated through rents in the mesentery,

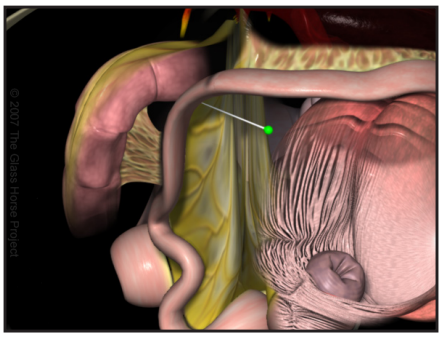

Figure 25. The ascending portion of the duodenum

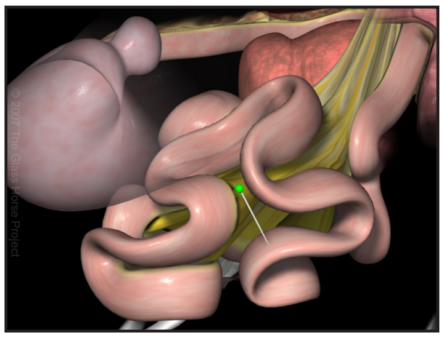

Figure 26. The jejunum lies in coils in the abdomen.

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through the inguinal canal, or through the epiploic foramen; these loops may also become twisted on the root of the mesentery.

The last part of the small intestine is the **ileum**, which empties into the cecum. The ileum is about 30 cm long, has a thick muscular wall, and terminates on the dorsomedial aspect of the cecum. A mesentery, called the **ileocecal fold**, attaches the ileum to the dorsal band of the cecum (Figure 27).

The small intestine is attached to the dorsal body wall by the great mesentery, which is comprised of the mesojejunum and mesoileum. The great mesentery contains the blood vessels, lymphatics, and nerves that supply the intestines.

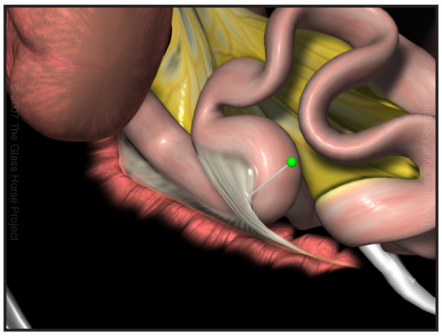

Figure 27. The ileum is attached to the cecum by the ileocecal fold.

Cecum

The **cecum** is a large comma-shaped fermentation vat that is positioned primarily to the right of the midline. In the average adult horse, the cecum is about 1 meter long and contains about 40 liters of ingesta and intestinal secretions. The cecum is comprised of three regions: the **base**, the **body**, and the **apex** (Figure 28). The base of the cecum is located dorsally in the right paralumbar fossa region. The base extends cranially to the costal portion of the diaphragm and the right lobe of the liver and is attached to the ventral surface of the right kidney, the root of the mesentery, and the pancreas. The body of the cecum extends from the base, resting on the ventral wall of the abdomen. The apex of the cecum rests on the abdominal floor and ends approximately 10 cm from the xyphoid cartilage.

The cecum has sacculations, otherwise known as **haustra**, and four **teniae** commonly called bands. The medial cecal band is covered by the medial cecal artery and vein, lymphatics, and lymph nodes; it joins the ventral band near the apex (Figure 29). The lateral cecal band is covered

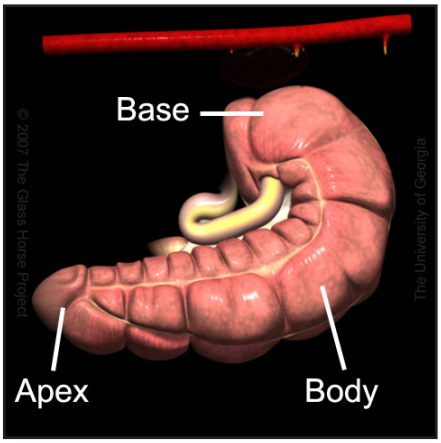

Figure 28. The cecum is comprised of the base, body, and apex.

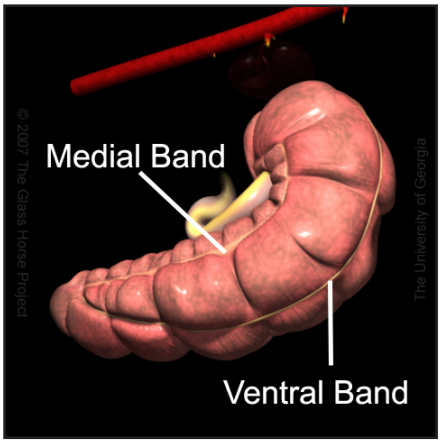

Figure 29. The medial band joins the ventral band near the apex.

by the lateral cecal artery and vein, lymphatics, and lymph nodes. The ventral cecal band, which is not covered by vessels, runs from the base toward the apex of the cecum and is the most easily palpated band per rectum (Figure 30). The ileocecal fold attaches to the dorsal band and is commonly used to locate the ileum during abdominal surgery (Figure 27; previous page). A strong triangular fold of tissue, the cecocolic fold attaches the lateral band of the cecum to the right ventral colon.

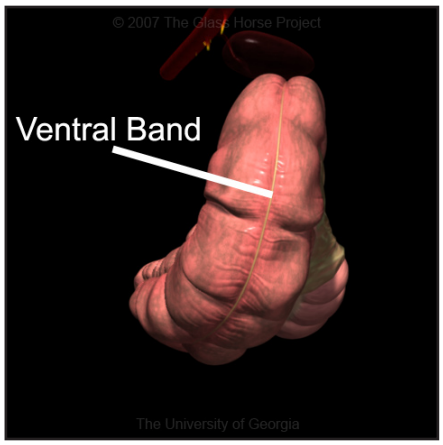

Figure 30. The ventral band is palpable during rectal examination.

Ascending or Large Colon

The **ascending colon**, or large colon, originates on the right side of the abdomen at its junction with the cecum, extends forward to the sternum, and continues around the left side of the abdomen to the pelvic region. At that point, the colon reverses its direction: the left dorsal colon extends forward to the diaphragm and then turns caudally to become the right dorsal colon. The right dorsal colon terminates in the short transverse colon, which is attached to the dorsal body wall near the cranial mesenteric artery. The **ascending mesocolon**, situated between the dorsal and ventral colons, extends from its point of origin at the root of the mesentery to the pelvic flexure.

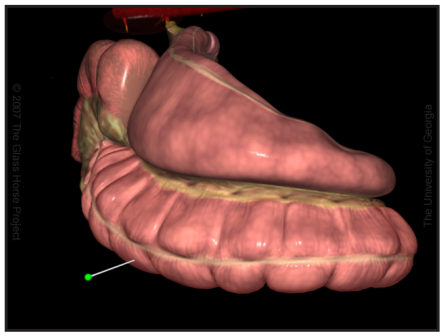

Figure 31. The right ventral colon has haustra and four bands.

The **right ventral colon** has a diameter of approximately 20 cm and is characterized by sacculations, haustra, and four longitudinal bands, two of which are incorporated into the mesocolic attachments (Figure 31). At about the level of the xyphoid cartilage of the sternum, the ventral colon bends sharply to the left side of the abdomen. This region of the ventral colon is called the **sternal flexure**. The **left ventral colon** has a diameter of approximately 20 to 25 cm and contains sacculations, haustra, and four bands (Figure 32). The medial and lateral free bands can be seen on the serosal surface of the colon. The other two bands are incorporated into the mesocolic attachments. Near the entrance to the pelvic inlet, the colon bends sharply dorsally, and its diameter decreases from about 20 cm to about 8 cm. This region, called the **pelvic flexure**, is a common site for impactions. From here, the left dorsal colon passes dorsal and often lateral to the left ventral colon. The **left dorsal colon** is not sacculated and has only one longitudinal band, located at the attachment of the mesocolon between the left dorsal and ventral colons. At about the level of the diaphragm and liver, the dorsal colon turns sharply toward the right side of the abdomen. This region of the dorsal colon is called the **diaphragmatic flexure**. The

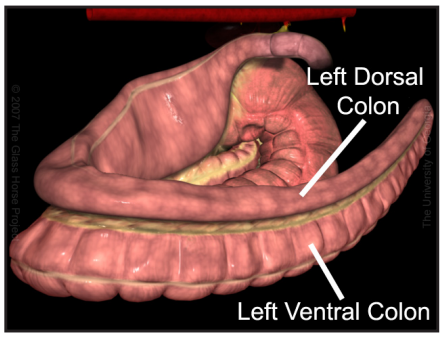

Figure 32. The left ventral and left dorsal colons on the horse's left side

right dorsal colon passes dorsal to the right ventral colon until it reaches the base of the cecum (Figure 33). As the right dorsal colon turns toward the left, its diameter decreases from a maximal size of about 40 cm to approximately 8 cm.

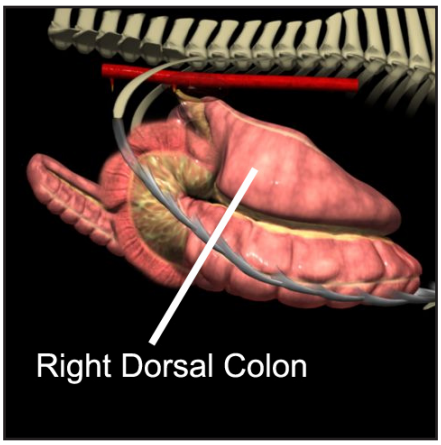

Figure 33. The right dorsal and right ventral colons on the horse's right side

Transverse Colon

The **transverse colon** is a short segment of intestine connecting the ascending and descending colons (Figure 34). It is positioned dorsally, cranial to the cranial mesenteric artery, and has a diameter of approximately 8 cm. It is attached to the dorsal aspect of the abdominal cavity by a very short transverse mesocolon.

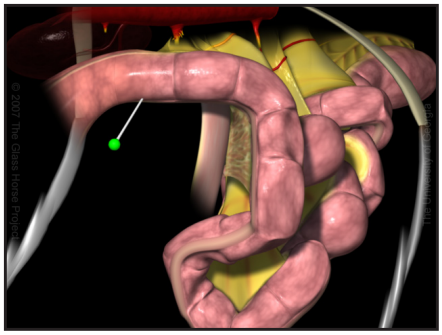

Figure 34. The transverse colon is cranial to the cranial mesenteric artery.

Descending or Small Colon

The **descending colon**, or small colon, is about 3 meters long, has a diameter of approximately 8 cm, contains sacculations and fecal balls, and has two longitudinal bands (Figure 35). One band is at the junction of the mesentery and the colon, and the other band is on the antimesenteric surface of the colon. The latter band is wide and easily palpable during a rectal exam. Characteristically, the mesentery of the descending colon contains a large amount of fat, making identification of the mesenteric vessels difficult.

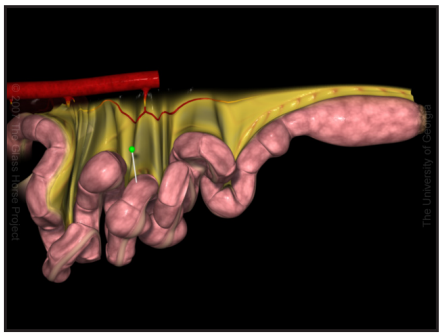

Figure 35. The descending colon has sacculations and two bands.

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Rectum

The **rectum** is approximately 25 cm in length, beginning at the pelvic inlet and terminating at the anus (Figure 36). Initially the rectum is covered by peritoneum and supported by the mesorectum. The distal portion of the rectum in the sacral region is retroperitoneal and is surrounded by connective tissue.

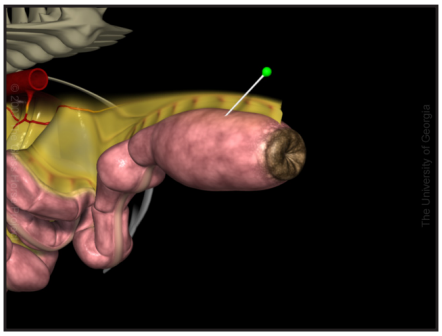

Figure 36. The rectum is positioned in the pelvic canal.

Liver

The horse's **liver** is situated adjacent to the diaphragm in the cranial aspect of the abdomen. It extends across the width of the abdomen and is divided into the left and right lobes, which account for the bulk of the organ, and the quadrate and caudate lobes. The **left lobe** is further subdivided into the left lateral lobe and the left medial lobe (Figure 37). The **left lateral lobe** is the thick portion of the liver to the left of a line connecting the esophageal impression to the fissure between the left and quadrate lobes. The left lateral lobe is easiest to identify when the liver is viewed from its parietal surface. The left medial lobe is the smaller of the two parts comprising the left lobe and is also visible when the liver is viewed from its parietal surface. The **right lobe**, which usually is undivided, sits primarily to the right of the caudal vena cava. The **quadrate lobe**, situated ventral to the caudal vena cava, extends across the midline (Figure 38). It is most evident when the liver is viewed from its parietal surface. The **caudate process**, which is the pointed extension of the caudate lobe of the liver, forms one of the boundaries of the **epiploic foramen**.

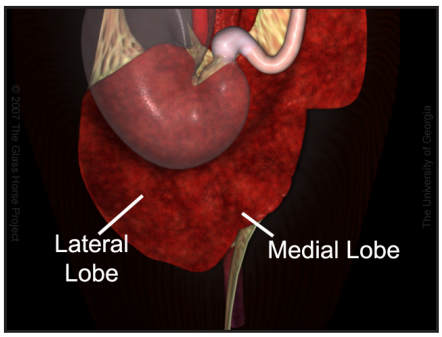

Figure 37. The liver's left lobe has left lateral and left medial lobes.

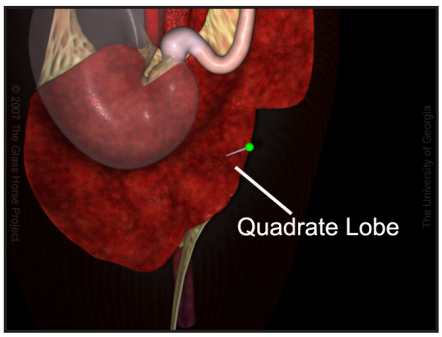

Figure 38. The liver's quadrate lobe extends across midline.

The liver has diaphragmatic and visceral surfaces. The **diaphragmatic surface**, which is convex and is positioned against the diaphragm, faces cranially and dorsally. In contrast, the **visceral surface** is concave and faces caudally and ventrally.

Blood from the intestines enters the liver via the portal vein, and bile is excreted into the duodenum via the **bile duct**. The **portal vein** is situated on the visceral surface of the liver just to the right of the division between the left and right lobes (Figure 39). The **caudal vena cava** runs along the dorsal aspect of the liver adjacent to the right lobe and dorsal to the quadrate lobe (Figure 40). The bile duct, also called the common hepatic duct, passes between two layers of mesoduodenum to enter the lumen of the duodenum approximately 15 cm from the pylorus.

The liver has several impressions or indentations due to close associations with other organs. The most obvious of these are the gastric impression, colic impression, and renal impression. The **gastric impression** is a concave area formed on the visceral surface of the liver by the adjacent stomach. The **colic impression** is a concave area that reflects contact of the liver with the diaphragmatic flexure and right dorsal colon. The **renal impression** is a concave area of the right lobe of the liver immediately adjacent to the right kidney.

The liver is connected to adjacent structures by a series of ligaments, the most notable of which are the right and left triangular ligaments and the coronary, falciform, and round ligaments (Figure 41). The **right triangular ligament** connects the dorsal aspect of the right lobe to the diaphragm, whereas the **left triangular ligament** connects the dorsal aspect of the left lobe to the diaphragm. The **coronary ligament**, which consists of two laminae, attaches the parietal surface of the liver to the diaphragm. The right and left laminae of the coronary ligament join below the vena cava to form the falciform ligament. The **falciform ligament** attaches the quadrate and left medial lobes of the liver to the diaphragm and ventral abdominal floor. This ligament can be recognized by its crescent shape and the presence of the round ligament running along its concave edge. The **round ligament** is the remnant of the umbilical vein, which carries blood from the placenta to the liver. In the adult horse, the round ligament is a fibrous cord along the concave edge of the falciform ligament.

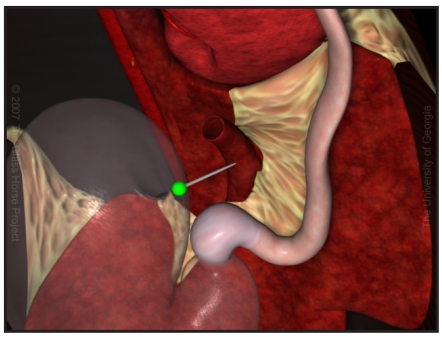

Figure 39. The portal vein enters the ventral aspect of the liver.

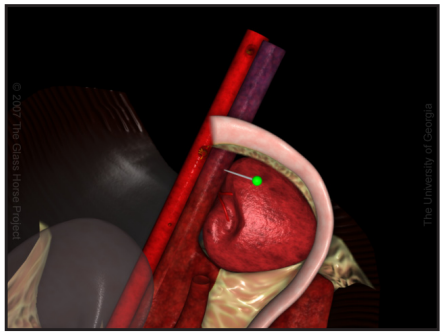

Figure 40. The caudal vena cava is adjacent to the liver's right lobe.

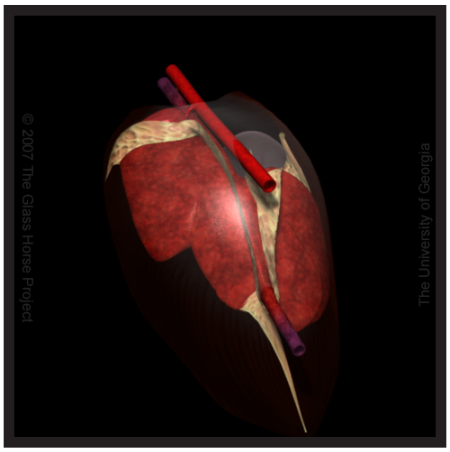

Figure 41. The ligaments that attach the liver to the diaphragm

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Spleen

The **spleen** is a large reservoir for red blood cells. It is situated on the left side of the abdomen, adjacent to the left body wall. The spleen is attached to the left kidney via the **renosplenic ligament** (Figure 42). The renosplenic ligament is the ventral part of the suspensory ligament of the spleen. It attaches the dorsomedial aspect of the spleen to the left kidney. The **cranial margin** of the spleen is concave and thin and is positioned between the greater curvature of the stomach and the diaphragm. The **caudal margin** of the spleen is convex and thin and can be palpated adjacent to the body wall during the rectal examination. The **hilus** is a groove on the medial aspect of the spleen in which the splenic vessels and nerves are positioned (Figure 43).

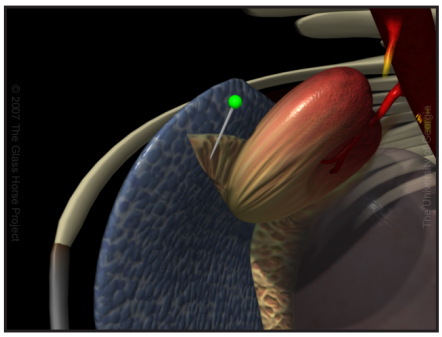

Figure 42. The renosplenic ligament connects the spleen and left kidney.

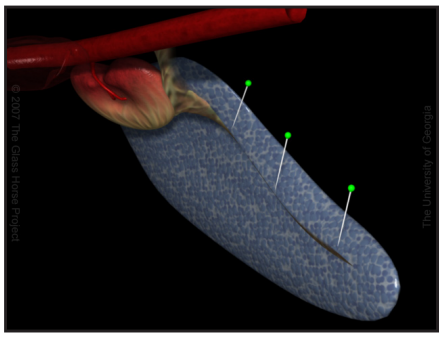

Figure 43. The hilus on the medial aspect of the spleen

Pancreas

The pancreas, which is comprised of a right lobe, body, and left lobe, is situated on the dorsal abdominal wall (Figure 44). The **right lobe** is positioned within the mesoduodenum and is approximately 15 cm in length. The **body** of the pancreas joins the two lobes at an angle of approximately 45 degrees, and cranially it is adjacent to the pyloric part of the stomach. Positioned within the deep leaf of the greater omentum, the **left lobe** is approximately 70% as long as the right lobe and is about 4 cm in width. The portal vein passes obliquely through the pancreas to reach the liver.

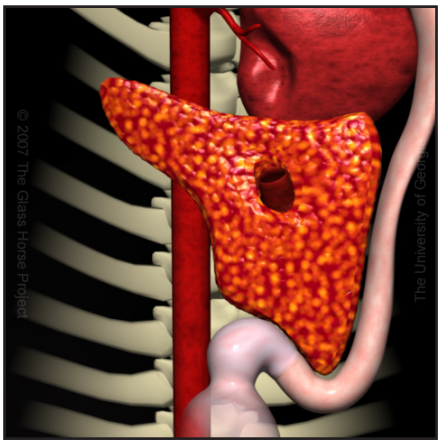

Figure 44. The pancreas is comprised of the right lobe, left lobe, and body.

Kidneys

The kidneys, which are part of the horse's urinary system, are retroperitoneal, meaning that they are not within the abdomen per se. The left kidney is positioned more caudally than the right kidney, and it hangs obliquely from the dorsal body wall (Figure 45). The left kidney and the spleen are joined by the **renosplenic ligament**, which forms the ventral border of the renosplenic space. The importance of this space will become obvious as you learn about the condition called left dorsal displacement of the large colon.

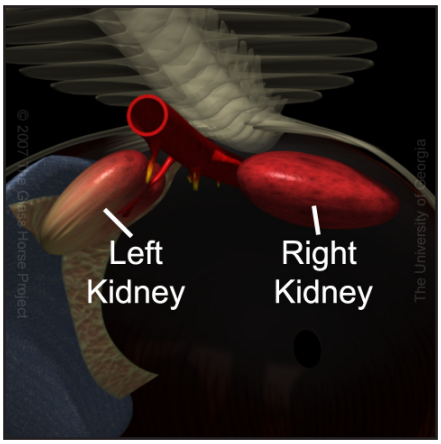

Figure 45. The left kidney hangs obliquely from the dorsal body wall.

Connecting Peritoneum

Some of the more difficult aspects of the abdomen to envision are the folds of peritoneum called mesenteries, ligaments, and omenta. **Mesenteries** connect the dorsal body wall to intestine, **ligaments** connect abdominal viscera to each other or to the body wall, and **omenta** connect other organs to the stomach.

On the horse's left side, peritoneum connects the diaphragm, spleen, and left kidney in the form of the **splenophrenic and renosplenic ligaments**; the peritoneum then continues to the stomach as the **gastrosplenic ligament** (Figure 46). In the cranial aspect of the abdomen, the **falciform, round, coronary, and triangular ligaments** connect the abdominal wall and diaphragm to the liver. The peritoneum continues from the liver to the stomach and duodenum in the form of the **lesser omentum** (Figure 47), which is comprised of the **hepatogastric and hepatoduodenal ligaments**. On the right side of the abdomen, peritoneum connects the dorsal body wall, small intestine, and colon in the form of the **mesoduodenum**, **great mesentery**, and **mesocolons**.

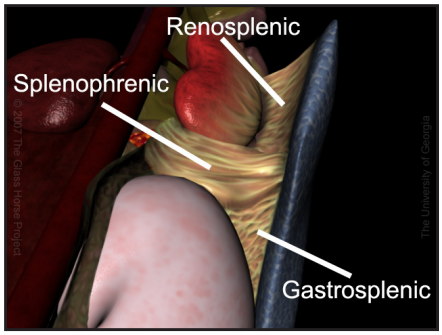

Figure 46. Ligaments connecting the spleen, kidney, and stomach

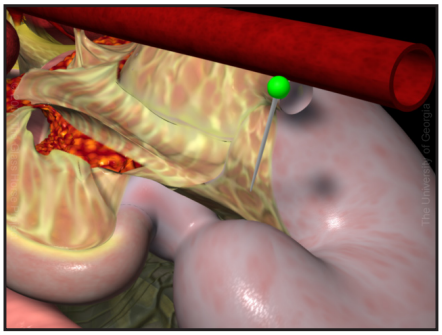

Figure 47. The lesser omentum attached to the stomach

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The **greater omentum** is a complex folding of peritoneum that engulfs the pancreas and connects the initial part of the descending colon, the transverse colon, the right dorsal colon, the duodenum, and the greater curvature of the stomach. Because it is continuous with the **gastrosplenic, splenophrenic, and renosplenic ligaments**, the greater omentum encompasses the caudal recess of the omental bursa (Figure 48). The omental bursa can be entered from the peritoneal cavity by passing through the **epiploic foramen**, which is bounded dorsally by the caudate process of the liver and the caudal vena cava and ventrally by the hepatoduodenal ligmament, portal vein, and pancreas (Figure 49). The epiploic foramen communicates with the vestibule of the omental bursa, cranially near the lesser curvature of the stomach as well as caudally with the much larger caudal recess of the omental bursa.

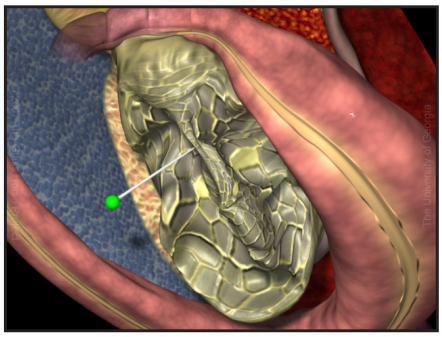

Figure 48. The greater omentum and omental bursa

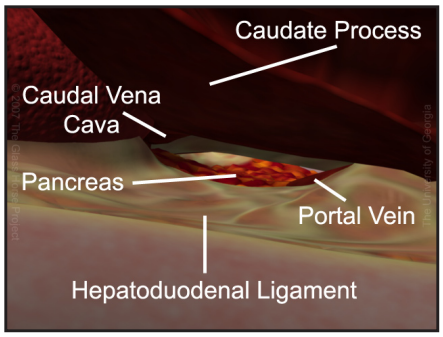

Figure 49. The epiploic foramen and its boundaries

Arteries

The horse's gastrointestinal tract is supplied by three arteries: the celiac, the cranial mesenteric, and the caudal mesenteric. The **celiac artery** primarily supplies the stomach, liver, spleen, pancreas, and initial part of the duodenum. The **cranial mesenteric artery** supplies the caudal portion of the duodenum, the jejunum, the ileum, the cecum, the ascending and transverse colons, and the initial part of the small colon. The **caudal mesenteric artery** supplies the rest of the small colon and the rectum.

The first branch of the celiac artery, the **splenic artery**, passes ventrally and to the left of midline. It provides pancreatic branches to the pancreas, splenic branches to the spleen, and the short gastric arteries and left gastroepiploic artery to the greater curvature of the stomach (Figure 50). The second branch, the **left gastric artery**, passes ventrally and cranially in the gastrophrenic ligament. It gives off branches to the

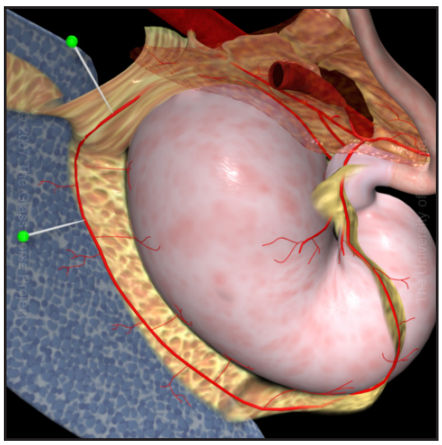

Figure 50. The splenic artery and its main branches

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distal part of the esophagus and then divides into branches that supply the parietal and visceral surfaces of the stomach (Figure 51).

The third branch of the celiac artery, the **hepatic artery**, passes cranially and ventrally on the surface of the pancreas, where it supplies part of that organ, and then it divides into left and right branches that supply the liver (Figure 52). The hepatic artery also gives off the **right gastric artery**, which supplies the pylorus and first part of the duodenum. The **gastroduodenal branch** of the hepatic artery splits into the **cranial pancreaticoduodenal artery**, which supplies part of the pancreas and the initial part of the duodenum, and the **right gastroepiploic artery**, which supplies the greater curvature of the stomach and forms an anastomotic arch with the **left gastroepiploic artery** (Figure 53).

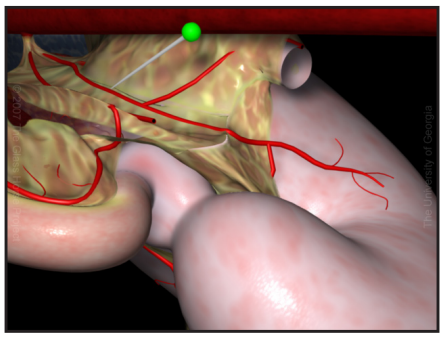

Figure 51. The left gastric artery and its branches

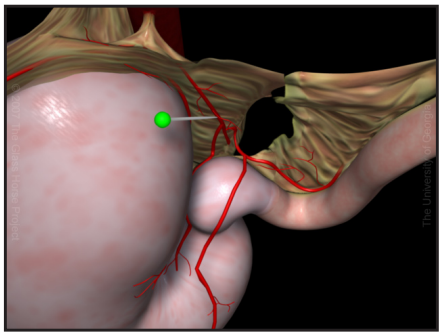

Figure 52. The hepatic artery and its branches

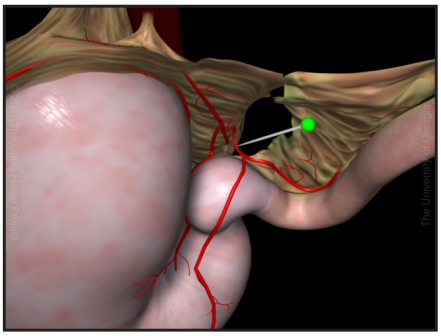

Figure 53. The gastroduodenal branch of the hepatic artery

One can also take the point of view of the organs in describing the branches of the celiac artery. According to this perspective, the stomach is supplied by the short gastric, left gastroepiploic, left gastric, right gastric, and right gastroepiploic arteries; the pancreas is supplied by branches from the splenic, cranial pancreaticoduodenal, and hepatic arteries; and the duodenum is supplied by the right gastric and cranial pancreaticoduodenal arteries that arise from the hepatic artery.

The second of the three main arteries supplying the horse's gastrointestinal tract is the **cranial mesenteric artery**. This artery itself is rather short. It gives off the caudal pancreaticoduodenal artery, numerous jejunal arteries, and a common trunk that splits into the right and middle colic arteries. The **caudal pancreaticoduodenal artery** supplies the distal half of the duodenum and anastomoses with the cranial pancreaticoduodenal artery. The **jejunal arteries** supply the jejunum, and the **right colic artery** supplies the entire dorsal colon (Figure 54). The **middle colic artery** supplies the transverse colon and the initial part of the descending colon. The cranial mesenteric artery continues distally as the **ileocolic artery** (Figure 55), which gives rise to the ileal artery, medial and lateral cecal arteries, and the colic branch. The ileal artery supplies the ileum, and the medial and lateral cecal arteries supply the medial and

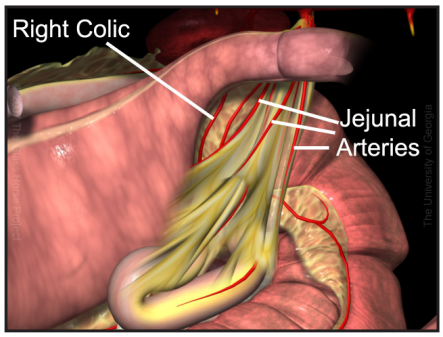

Figure 54. The jejunal arteries and right colic artery

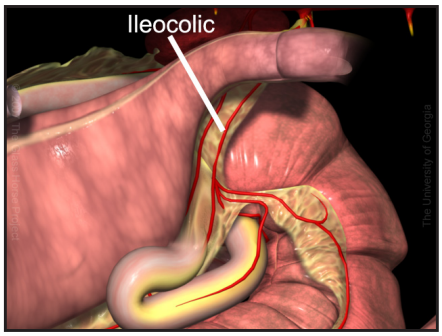

Figure 55. The ileocolic artery and its branches

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lateral aspects of the cecum (Figure 56). The colic branch supplies the entire ventral colon and anastomoses with the right colic artery at the pelvic flexure (Figure 57).

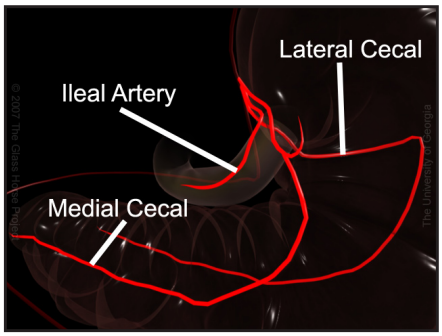

Figure 56. The ileal, lateral, and medial cecal arteries

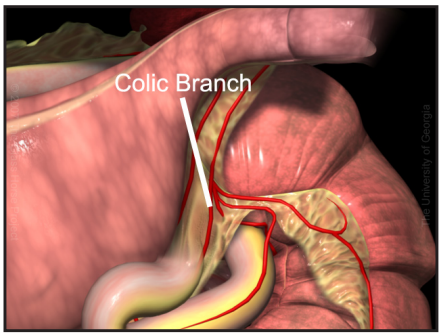

Figure 57. The colic branch

Once again, one can describe this artery and its branches from the point of view of the organs that it supplies. The duodenum receives part of its blood supply from the caudal pancreaticoduodenal artery. The jejunum and ileum receive their blood supply from the jejunal and ileal arteries. The cecum is supplied by the medial and lateral cecal arteries, and the ascending colon is supplied by the right colic artery and colic branch. Finally, the transverse colon and proximal descending colon are supplied by the middle colic artery.

The third major artery supplying the gastrointestinal tract, the **caudal mesenteric artery**, splits into two branches, the left colic and cranial rectal arteries (Figure 58). The left colic artery supplies the majority of the descending colon, whereas the cranial rectal artery supplies the rectum. The caudal mesenteric artery supplies the descending colon and rectum.

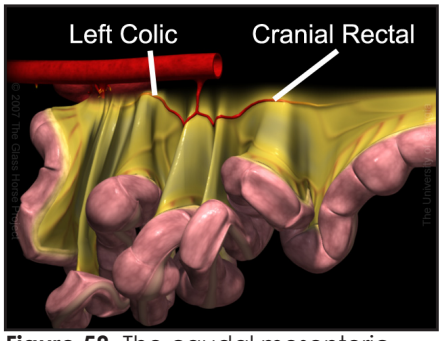

Figure 58. The caudal mesenteric artery and its branches

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Categories of Disease

Obstruction

An obstruction occurs when the normal movement of ingesta is restricted or prevented but no change occurs in the blood supply to the intestine. Most often obstructions occur when ingesta fails to move from a portion of the bowel having a large diameter into a portion with a smaller diameter (Figures 59a and 59b). Very often the bowel proximal to the obstruction distends with gas. Examples of obstructions include impaction of the large colon at the pelvic flexure, enterolithiasis, and adhesions involving the small intestine.

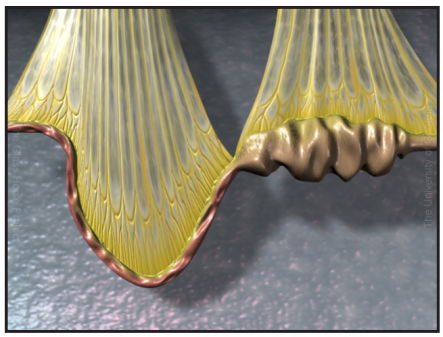

Figure 59a. A simplified model of a normal gastrointestinal tract

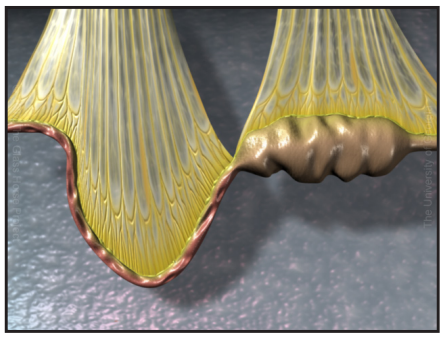

Figure 59b. Obstruction of the bowel and distention proximally

Distention

Distention of the intestine occurs when excess gas in the intestinal lumen stretches the wall of the intestine (Figures 60a and 60b). When the stomach is involved, the condition is called dilation; when the cecum or colon is involved, it is referred to as tympany. The most common examples of distention are cecal tympany and gastric dilation.

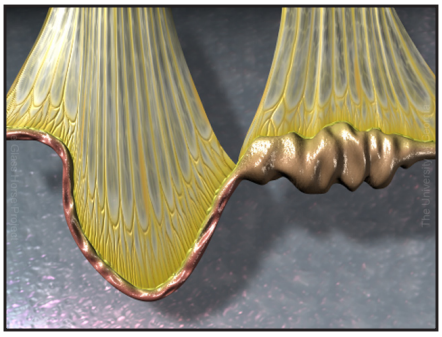

Figure 60a. A simplified model of a normal gastrointestinal tract

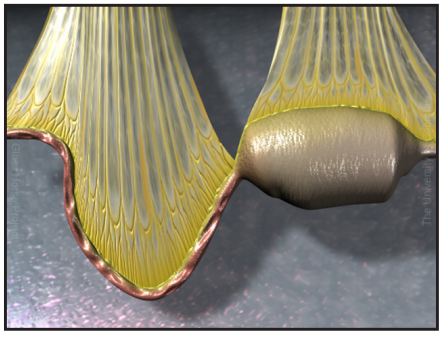

Figure 60b. Distention of the intestine

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Spasm

Normally, contractions of smooth muscle cells in the wall of the intestine occur in a well-coordinated manner, moving the ingesta aborally along the gastrointestinal tract. In contrast, abnormal, uncoordinated contractions (Figures 61a and 61b), otherwise known as spasms, may cause a horse to feel abdominal pain. In these instances, the blood supply to the intestine is normal, and there is no obstruction to the movement of ingesta. Presumably, spasms may occur either in the small intestine or the large colon.

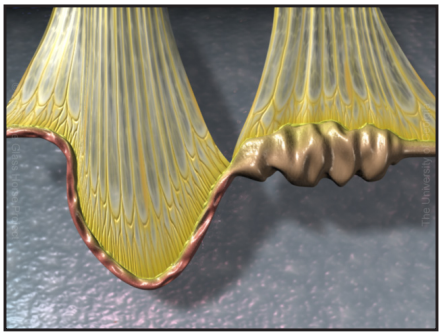

Figure 61a. A simplified model of a normal gastrointestinal tract

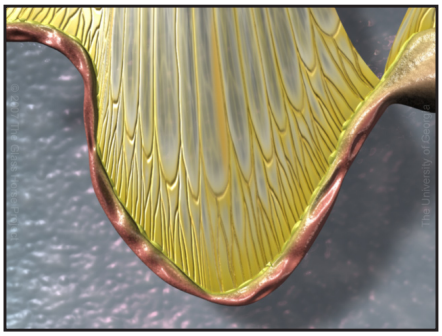

Figure 61b. Spasms of intestinal muscles cause pain.

Strangulation Obstruction

Strangulation obstructions occur when both the flow of ingesta and the intestinal blood supply are interrupted. This can occur if the intestine moves through an opening, such as a tear in the mesentery, or if the intestine twists enough to occlude the lumen and the vessels (Figures 62a and 62b). The affected intestine becomes edematous and ischemic, and the intestine proximal to the lesion distends (Figure 62c). Examples of strangulation obstructions include large colon volvulus, inguinal hernia, and incarceration of small intestine through a mesenteric rent.

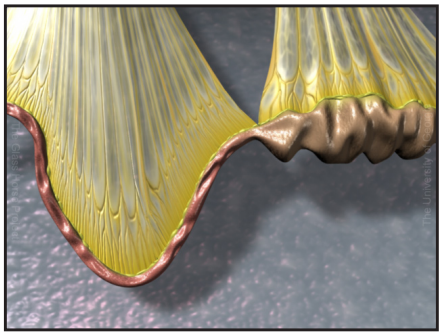

Figure 62a. A simplified model of a normal gastrointestinal tract

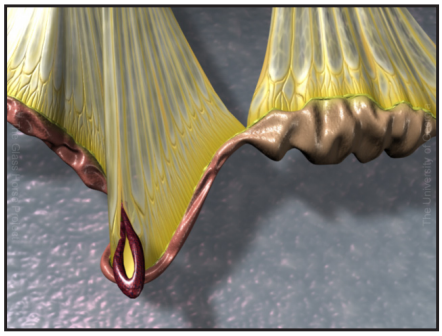

Figure 62b. Strangulation obstruction of a section of intestine

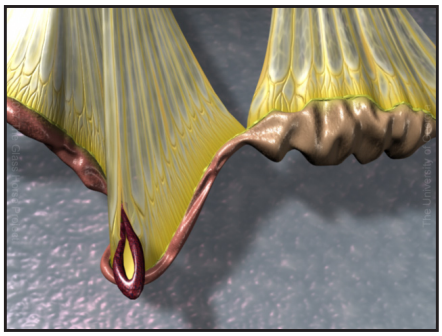

Figure 62c. Distention of intestine proximal to the strangulation

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Ulceration

Normally, a layer of mucosal epithelial cells covers the interior of the intestine. Loss of this layer down to the submucosa is referred to as ulceration (Figures 63a and 63b). This may result in bleeding into the intestinal lumen and even perforation of the intestinal wall. The most common examples of conditions involving ulceration are gastric ulcer disease, which occurs in the stomach, and right dorsal colitis, which occurs in the right dorsal colon.

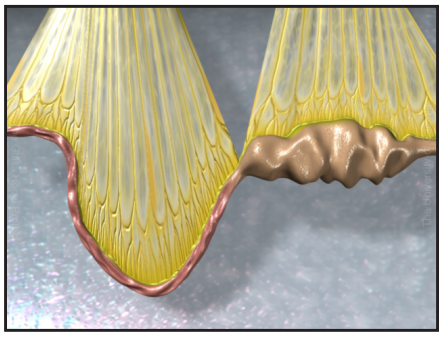

Figure 63a. A simplified model of a normal gastrointestinal tract

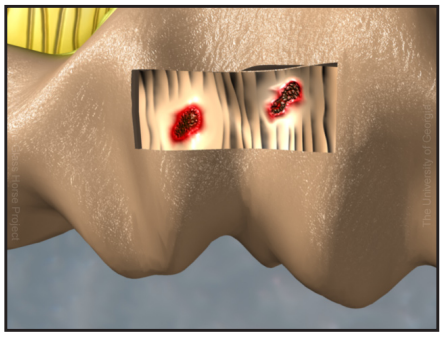

Figure 63b. Ulceration of the mucosa

Enteritis and Colitis

Enteritis refers to inflammation of the small intestine. This inflammation results in thickening of the intestinal wall, secretion of fluid into the intestinal lumen, and distention of the intestine with gas and fluid (Figures 64a and 64b). Colitis refers to inflammation of the colon. The inflamed colonic wall becomes edematous, and large volumes of fluid are secreted into the colonic lumen (Figure 64c). Although proximal enteritis is the only clinical disease that results in enteritis in adult horses, there are numerous causes for colitis, such as salmonellosis and clostridial enterocolitis.

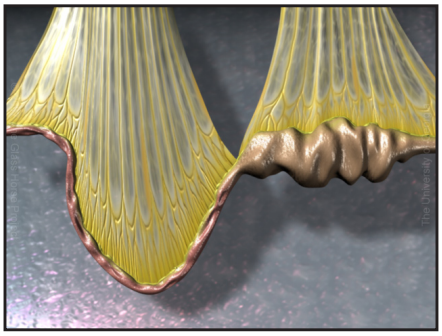

Figure 64a. A simplified model of a normal gastrointestinal tract

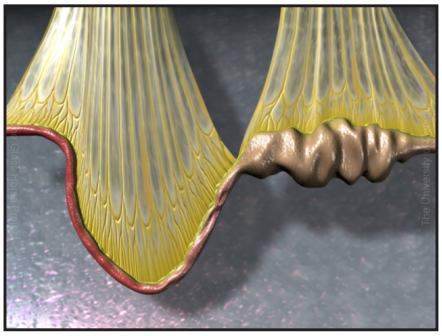

Figure 64b. Inflammation and distention of the small intestine

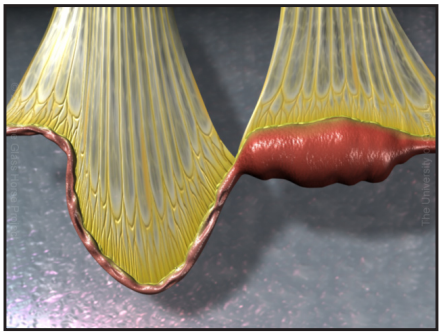

Figure 64c. Inflammation and distention of the colon

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Peritonitis

Inflammation of the lining of the peritoneal cavity is called peritonitis. Peritonitis commonly occurs secondary to strangulated or severely inflamed intestine and results in the movement of large numbers of white blood cells into the peritoneal cavity (Figures 65a and 65b). Examples of conditions that cause peritonitis include strangulation obstruction of the small intestine by a pedunculated lipoma and perforation of the intestine during an abdominal paracentesis.

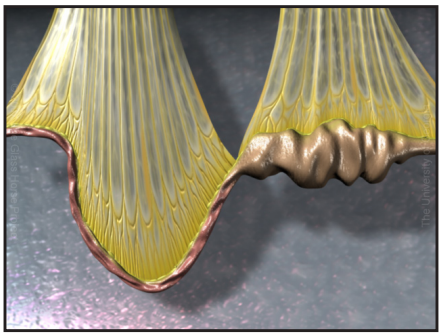

Figure 65a. A simplified model of a normal gastrointestinal tract

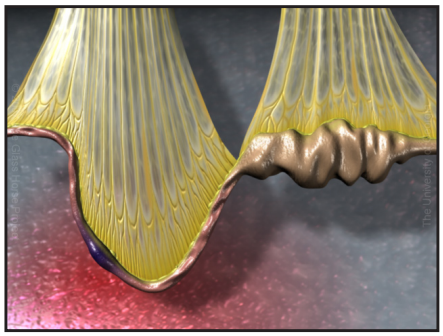

Figure 65b. Inflammation of the peritoneum secondary to ischemia

Nonstrangulating Infarction

Loss of blood supply to part of the intestine in the absence of a displacement or incarceration is called a nonstrangulating infarction (Figures 66a and 66b). When this occurs, the affected tissue becomes ischemic. This condition may affect the small intestine, cecum, or colon, and it may simultaneously affect more than one of these regions of the intestine.

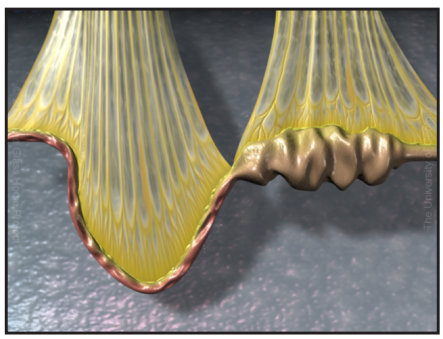

Figure 66a. A simplified model of a normal gastrointestinal tract

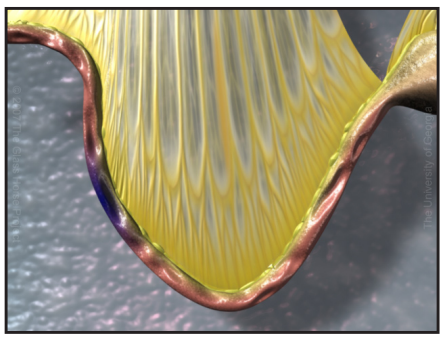

Figure 66b. Ischemia of the intestine due to loss of blood supply

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Diagnostic Approach

Clinical Signs of Colic

Horses with colic often exhibit one or more clinical signs that can be interpreted as evidence of pain originating within the abdomen. However, not all horses with colic exhibit all of these clinical signs, nor do all horses with the same disease show the same signs. While this section explains the most common clinical signs associated with abdominal pain, it does not include them all.

One of the most common signs of abdominal pain is repeated **pawing** with a front foot (Figure 67). The horse's head typically is near the ground, and the horse paws repeatedly with one fore foot or the other. Some horses may quit for a short time and then start pawing again.

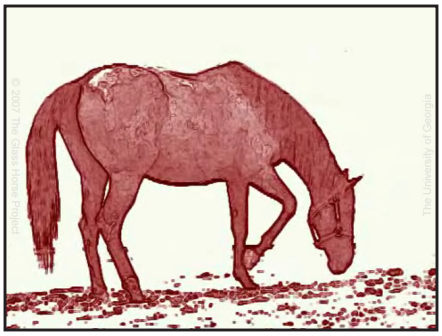

Figure 67. Pawing with a forefoot

Many horses **stretch out** in response to abdominal pain. They often raise their heads, plant their forefeet out in front of them, and then lean backwards (Figure 68). Presumably they do this to try to relieve excessive pressure or tightness they feel in the abdomen. Years ago people said horses that stretched out had "kidney colic," but the stretching has nothing to do with kidney problems.

Figure 68. Stretching of the head, neck, and back

Another typical sign of abdominal pain is turning to look at the **flank** (Figure 69). Very often horses showing this sign stand stretched out and then turn to look at the abdomen.

Often horses with colic dance around, curl up their legs, and flop onto their bellies, and many will stand back up, turn around, and then flop down again. Many horses will look at their flanks when they are in the down position (Figure 70).

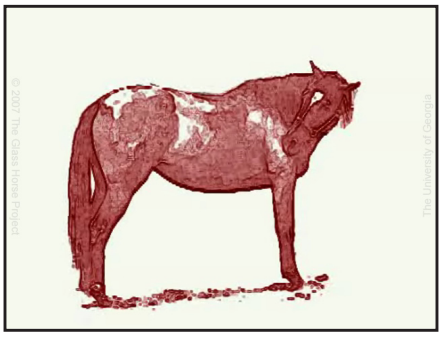

Figure 69. Flank watching

A sign commonly associated with more severe diseases is **rolling** (Figure 71). Horses that are in continuous pain will do this repeatedly and can hurt themselves or their handlers in the process. Years ago people thought that horses twisted their intestines when they rolled, but it is far more likely that they roll because they already have a twist. However, many horses without intestinal twists also show this sign.

Figure 70. Lying down due to abdominal pain

Figure 71. Rolling due to abdominal pain

It is important to recognize that some horses are more stoic than others and that some horses will cease showing signs of pain while they are being handled. Veterinarians must keep these facts in mind when examining horses with colic, and many will choose to observe a horse set free in its stall to see if it still shows signs of abdominal pain.

Examination of a Horse with Colic

This exam assesses the cardiovascular, gastrointestinal, and pulmonary systems. The cardiovascular system is evaluated by determining the heart rate, feeling the strength of the pulse, and examining the oral mucous membranes. The heart rate is determined most easily by listening to the heart with a stethoscope. **Heart rate** increases due to pain, excitement, and hypovolemia and as part of the systemic inflammatory response to bacterial toxins that enter the bloodstream in horses with severe gastrointestinal diseases. The strength of the **pulse** in the facial artery is assessed subjectively (Figure 72), recognizing that a weak, thready pulse is associated with poor cardiovascular function due to circulatory shock. In the final part of the cardiovascular assessment, the oral mucous membranes are examined for color, moistness, and capillary refill time. Mucous membranes in a horse with adequate cardiovascular function are pink and moist. The **capillary refill time** is determined by applying pressure with a finger or thumb on the gingiva, which causes blanching of the tissue, and then counting the time it takes for perfusion to return (Figures 73 and 74). Horses with adequate cardiovascular function have a capillary refill time of 1 to 2 seconds.

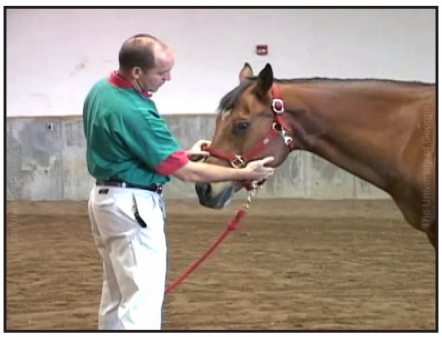

Figure 72. Palpation of the pulse in the facial artery

In the gastrointestinal portion of the examination, the horse's abdomen should be **ausculted** over several areas to determine the frequency and strength of intestinal sounds that are associated with intestinal muscle function. The abdomen should be ausculted over four sites: dorsally and ventrally on both the right and left sides. Ventrally on either side, sounds char-

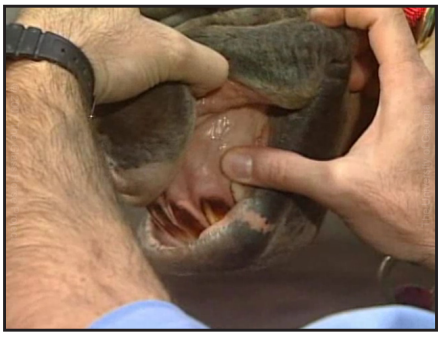

Figure 73. Applying pressure to the gingiva

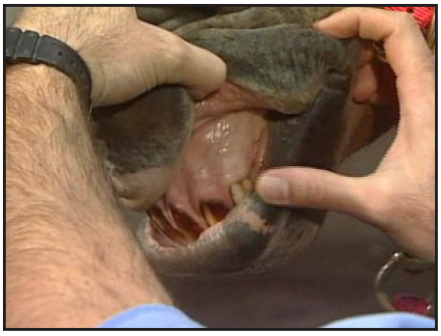

Figure 74. Release of pressure from the gingiva

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acteristic of colonic mixing contractions should be heard approximately 2 to 4 times per minute. Very often, longer gurgling sounds can be heard over the cecum on the right side;these sounds reflect emptying of ileal contents into the cecum as well as the vigorous mixing of cecal contents (Figure 75). An absence of intestinal sounds is consistent with severe intestinal problems that may require surgery.

Percussion of the horse's abdomen with a stethoscope is used to identify gas-filled portions of the intestine. When tapping on the side of the abdomen, the location of high-pitched sounds can be used to determine the relative size and location of the affected intestine. Generally, high-pitched sounds heard on percussion over the right paralumbar fossa are due to **gas distention** of the cecum, whereas the same findings over the left paralumbar fossa are due to distention of the large colon (Figure 76).

In the pulmonary assessment, recording the horse's respiratory rate and breathing pattern is important. The rate may increase due to pain, because of increased abdominal pressure against the diaphragm, and in response to metabolic acidosis. Short, quick, or labored breaths are also an indication of pain or excessive pressure on the diaphragm. In rare cases it may be possible to diagnose a diaphragmatic hernia by ausculting the thorax.

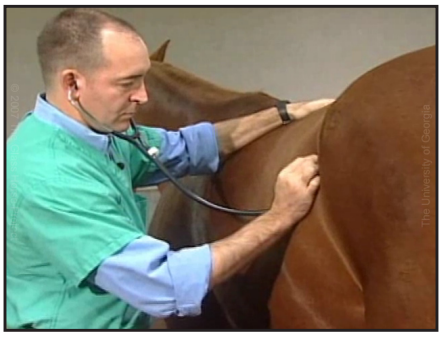

Figure 75. Auscultation of the abdomen

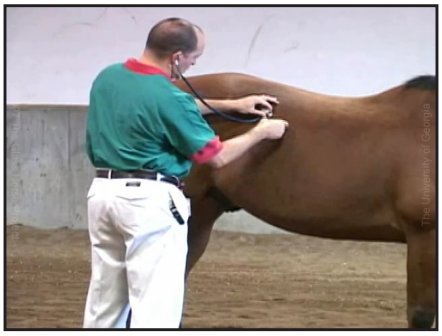

Figure 76. Percussion of the abdomen

Passing the Stomach Tube

In some diseases, the horse's stomach may become distended with gas or fluid, causing the horse to be painful. If the pressure is not relieved, the stomach can rupture. To determine if there is excessive gas or fluid present, the veterinarian passes a tube along the **ventral aspect** of the nasal passage (Figure 77), through the pharynx, into the esophagus, and into the stomach.

Although the end of the tube usually initiates a swallowing reflex when it touches the horse's larynx, the veterinarian may need to move the tube back and forth to initiate this reflex. When the horse swallows the tube, it is passed into the esophagus, which is positioned on the left side of the trachea. The veterinarian can ensure that the tube is in the esophagus by palpating the end of the tube as it moves down the neck (Figure 78). Once the tube reaches the stomach, its position is confirmed by blowing into the tube to allow the release of gas having the characteristic smell of gastric contents.

If the stomach is emptying its contents normally into the small intestine, pumping a few liters of water from a bucket into the stomach and then lowering the end of the tube to create a siphon results in retrieval of approximately the same total volume of fluid. If the horse's stomach is distended due to excessive production of gas, passing the stomach tube

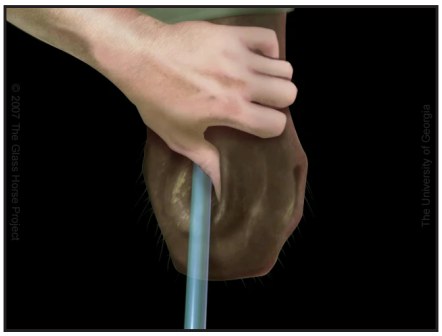

Figure 77. Passing the stomach tube on the ventral aspect of the nares.

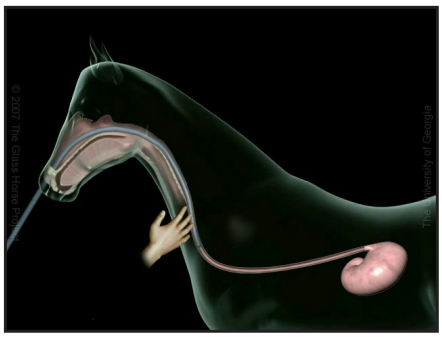

Figure 78. Palpating the stomach tube in the esophagus

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will result in rapid expulsion of the gas and elimination of the pain. If the horse's stomach is distended due to accumulation of fluid from an obstructed or inflamed small intestine, pumping a few liters of water into the stomach and developing a siphon may result in retrieval of a much larger volume of fluid, called **gastric reflux** (Figure 79). Horses with gastric reflux frequently require intensive care, and many need emergency abdominal surgery to treat the underlying problem.

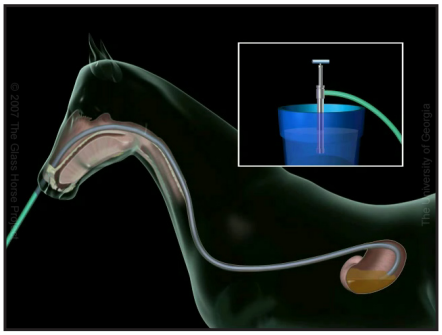

Figure 79. Developing a siphon to remove gastric reflux

Rectal Examination

One of the most important parts of the diagnostic work up of a horse with colic is the rectal examination. To detect abnormalities during a rectal examination the examiner must have an understanding of the position and feel of the normal anatomy.

A systematic examination is necessary to ensure all structures in the abdomen are palpated. After appropriate sedation and restraint of the horse, the examiner empties the rectum of fecal material and advances the hand into the center of the abdomen. Often, pausing from rectal manipulation for a few seconds allows the horse to relax.

Next, the examiner's hand is advanced to the left dorsal quadrant of the abdomen to find the caudal edge of the **spleen** (Figure 80). The edge of the spleen can be moved and has a characteristic bumpy surface. Depending on the size of the horse, advancing the hand cranially along the medial edge of the spleen reveals the renosplenic space, also called the nephrosplenic space. The **renosplenic ligament** forms a shelf between the spleen and the left kidney. By moving the hand medially, the posterior pole of the left kidney can be felt as a firm mass attached to the dorsal body wall. In large horses the examiner may not be able to touch the kidney.

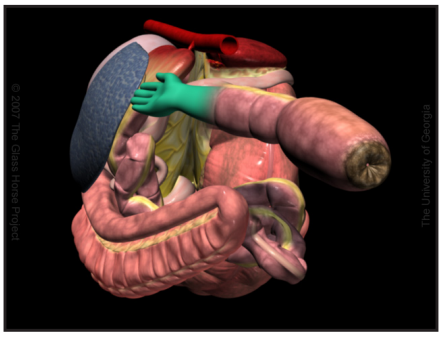

Figure 80. Palpation of the caudal edge of the spleen

The examiner's hand is then moved dorsally and towards the midline to locate the aorta, which can be identified by feeling a strong pulsation with each heartbeat. Following the aorta cranially, the **root of the mesentery** can be felt as a sheet of tissue, and in some horses a pulse can be felt in the cranial mesenteric artery as it courses ventrally within the mesentery (Figure 81).

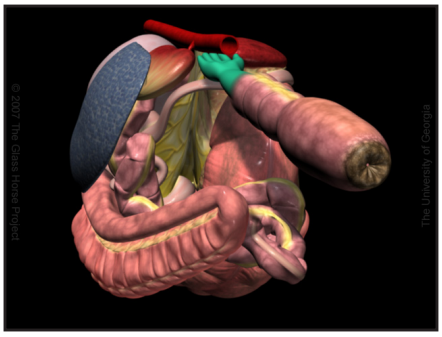

Figure 81. Palpation of the root of the mesentery

The hand is then moved to the right dorsal quadrant over the base of the cecum. Although the duodenum is in this region, it cannot be identified in the normal horse because it is flaccid. At this point, the examiner's hand is moved ventrally over the base of the **cecum**, allowing recognition of the ventral taenia (also known as the **ventral cecal band**), a thin, fibrous band that courses ventrally and toward the midline (Figure 82). Because the cecum usually contains a relatively small volume of watery ingesta, it is normally flaccid. By sweeping the hand to the medial surface of the cecum,

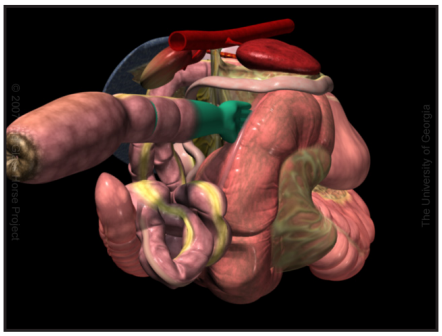

Figure 82. The ventral cecal band is palpated to the right of midline.

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it is sometimes possible to palpate the medial cecal band and pull it caudally to increase tension on the cecum. The cecal bands are followed to the right ventral quadrant of the abdomen.

From here, the examiner's hand is moved along the ventral body wall to the left ventral quadrant. Although fecal balls may be identified in the **small colon** in more than one quadrant, they are often found in the left ventral quadrant. Palpation of the left portion of the large colon is often possible by moving the hand cranially from the pelvic brim in the left ventral quadrant (Figure 83). In some instances, it is not possible to find this portion of the colon, either because it is positioned too far cranially or because its consistency does not allow palpation of a distinct structure. The left dorsal colon has a smooth serosal surface, whereas the left ventral colon has three bands that can sometimes be palpated.

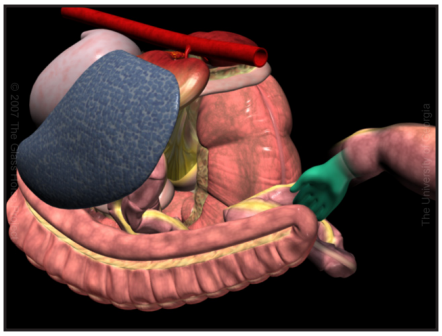

Figure 83. Palpation of the left ventral and left dorsal colons

Because it is flaccid and rarely filled with ingesta, small intestine normally cannot be identified by rectal palpation. If, however, the small intestine is distended or thickened, it can often be detected in the center of the abdomen between the left and right ventral quadrants.

As the hand is withdrawn from the small colon into the rectum, the urogenital structures should be examined. These include the inguinal rings, just lateral to each side of the pelvic brim in the stallion, the uterus and broad ligaments in the mare, and the bladder.

Abdominal Paracentesis

Another important diagnostic procedure, the abdominal paracentesis is performed at the lowest point of the abdomen either on the midline or slightly to the right. After the hair is clipped and a surgical prep is completed, a small bleb of local anesthetic is injected subcutaneously to desensitize the area. Using a #15 scalpel blade, a small stab incision is made through the skin and subcutaneous tissue and into, but not through, the linea alba (Figure 84). The blunt end of a sterile cannula is then seated in this partial incision in the linea alba, and with upward pressure the point of the cannula is inserted into the peritoneal cavity. In doing so, the cannula passes through the linea alba, retroperitoneal fat, and peritoneum (Figure 85).

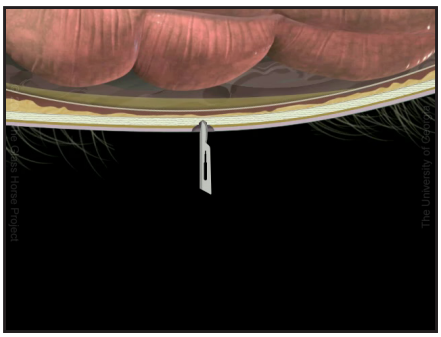

Figure 84. Incising the skin and subcutaneous tissue on ventral midline

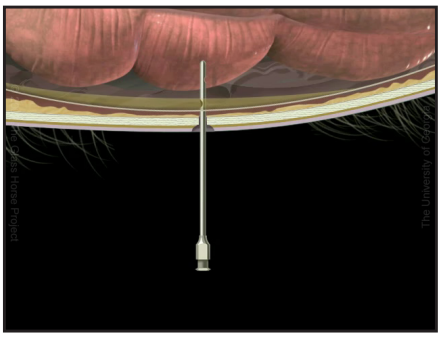

Figure 85. Insertion of the sterile cannula into the peritoneal cavity

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With some gentle maneuvering of the cannula, peritoneal fluid enters the holes in the cannula and flows by gravity into a sterile tube containing EDTA (Figure 86). Normal peritoneal fluid is clear and pale yellow, contains fewer than 5,000 cells per microliter, and has a protein concentration of less than 2 g/dl; it is classified as a transudate. Microscopic examination of normal peritoneal fluid reveals a mixture of neutrophils and mononuclear cells.

Based on the protein concentration and the total white blood cell count, abnormal samples are classified as either modified transudates or exudates. A **modified transudate** has either an increased concentration of white blood cells, most commonly neutrophils and large mononuclear cells, or an increased protein concentration. Peritoneal fluid is classified as an **exudate** if both the total white blood cell concentration and the protein concentration are increased.

Typically, peritoneal fluid is normal in horses with intestinal obstructions unless the duration of the obstruction is prolonged. In those instances, the fluid may become turbid as the number of white blood cells and concentration of protein increase.

If there is ischemic or inflamed intestine in the abdomen, the peritoneal fluid becomes **serosanguinous**, meaning that it is turbid and/or blood tinged (Figure 87). Serosanguinous fluid samples vary from red to brown in color and contain increased numbers of erythrocytes; the hematocrit of the fluid is usually less than 1 to 2%.

In addition to the serosanguinous fluid that occurs in horses with ischemic or inflamed intestine, the peritoneal fluid may be bloody under three other circumstances. First, the horse may have **intra-abdominal hemorrhage** either from an injured or ruptured blood vessel or from an organ, such as the liver, spleen, or kidney. This condition is called **hemoabdomen or hemoperitoneum**. If the hemorrhage is severe, the horse may show signs of shock. Generally, the hematocrit and total protein concentration in the peripheral blood will decrease over time, whereas the hematocrit of the peritoneal fluid will increase. Microscopic examination of this fluid often reveals phagocytosis of erythrocytes by the white blood cells.

Second, the blood may have come from an inadvertent **puncture of the spleen** (Figure 88). Because one of the spleen's functions is to store red blood cells, the hematocrit of a sample contaminated by splenic blood exceeds that of the peripheral blood. Microscopic examination of the fluid may reveal the presence of erythroid precursor cells as well as an increased number of small lymphocytes and platelets.

Third, the blood may originate from a **small vessel in the skin** or body wall that was damaged during the paracentesis (Figure 89). When this occurs, the peritoneal fluid from the cannula will initially be streaked with blood but may intermittently return to normal depending on the position of the cannula. It may be necessary to reposition the cannula to obtain a representative sample of peritoneal fluid.

Abdominal fluid from horses with **peritonitis** secondary to an infection is cloudy, and the white blood cell count often exceeds 100,000 cells per mi-

Figure 86. Collection of peritoneal fluid

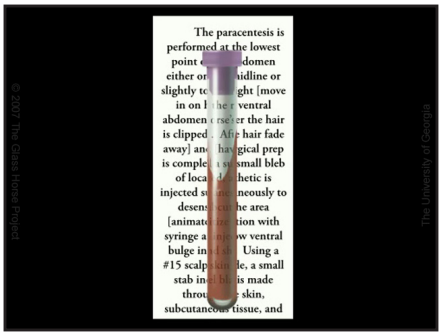

Figure 87. Serosanguinous peritoneal fluid

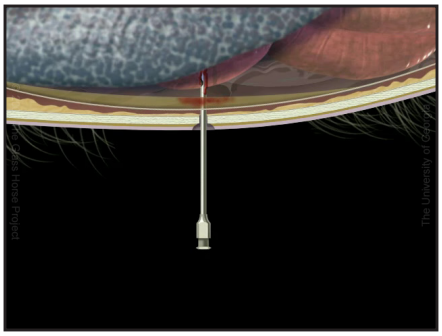

Figure 88. Puncture of the spleen during abdominal paracentesis

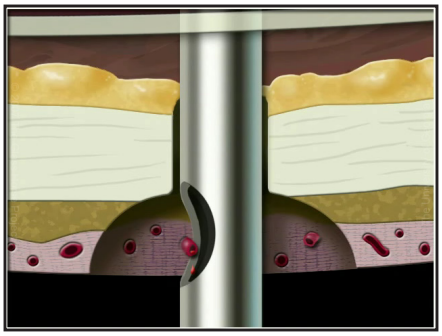

Figure 89. Contamination of peritoneal fluid with blood from a small vessel

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croliter. Bacteria may be seen both intracellularly and extracellularly when infection is present (Figure 90). When the bacteria appear morphologically homogeneous, the source may be an intra-abdominal abscess. When there is a mixed population of bacteria, they are more likely present due to compromised intestine. The presence of **degenerative neutrophils**, characterized by swollen, pale-staining, poorly segmented nuclei, is a sign of infection, even when bacteria are not seen. If infection is suspected, peritoneal fluid should be submitted for bacterial culture and antimicrobial sensitivity testing.

Whenever peritoneal fluid appears turbid and greenish brown and is malodorous, it is critical to determine whether the fluid was the result of an **enterocentesis** or a ruptured or leaking segment of intestine. Microscopic examination of the fluid may help distinguish these conditions. Although fluid samples from both an enterocentesis and a ruptured intestine often have a pleomorphic population of bacteria, protozoa, and plant material, fluid from an enterocentesis should have very few leukocytes (Figure 91). In some cases, the leukocyte count may be spuriously high as electronic cell counters

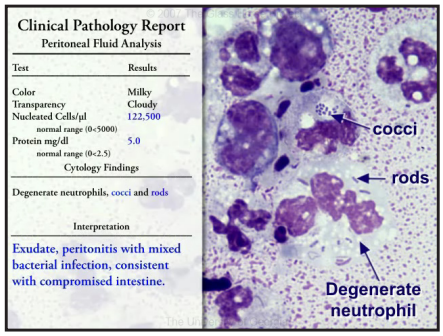

Figure 90. Intracellular and extracellular bacteria

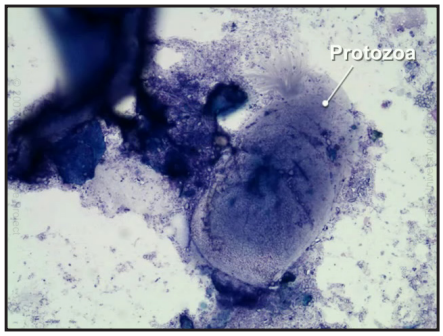

Figure 91. An example of fluid obtained by enterocentesis

often will count clumps of bacteria and plant material. If the intestine has ruptured, with time leukocytes will appear in the fluid and frequently will contain intracellular bacteria and/or appear degenerative.

Information gained by performing an abdominal paracentesis is of the most use if the fluid reflects the condition of the intestines. Peritoneal fluid may appear normal early in the development of a strangulating lesion or if the abnormality is separated from the paracentesis site. In the latter instance, the fluid obtained can provide misleading information. As is true for the other diagnostic procedures, information provided by an abdominal paracentesis must be considered in context with all other examination findings.

Abdominal Ultrasonography

Orientation of the Ultrasound Beam

Ultrasound images are generated when ultrasound waves are transmitted from the surface of a transducer, pass into the tissues, and are reflected back to a sensor. There are several different types of ultrasound transducers, but most imaging of the horse's abdomen is done with a **2 to 5 MHz curvilinear array transducer**. With the curvilinear transducer, ultrasound waves radiate from a curved footprint at the point of contact on the patient and generate a "pie-shaped" image through the plane of projection into the horse.

Although high frequency ultrasound waves produce images with excellent resolution, they do not penetrate tissues to the depth often needed to evaluate the equine abdomen. In years past, low frequency transducers were used for this purpose. Fortunately, most of today's tranducers produce waves of a range of frequencies, allowing the ultrasonographer to generate the best image by selecting the appropriate depth of penetration of the beam. In this example, an image of the horse's spleen and left kidney was obtained via the 16th intercostal space on the horse's left side (Figure 92). Initially, the image on the screen includes the skin, spleen, and kidney; note the depth on the scale to the right. The portions of the image at the **top of the screen** reflect those areas of the anatomy **closest to the transducer**, adjacent to the area of contact between the transducer and the skin (Figure 93). Portions of the image at the bottom of the screen reflect the tissues deeper within the abdomen. By dialing back the depth of penetration, the kidney and deeper structures can be eliminated, thereby filling the screen with the skin, intercostal muscles, and spleen (Figure 94).

Ultrasound transducers have an **orientation marker** that orients the image obtained from the patient, allowing the machine to project the image in a consistent manner on the screen. Most imaging is performed with this orientation marker at the **12 o'clock position** or towards the **dorsal aspect** of the patient (Figure 95). When the image is displayed on the screen, most ultrasound machines add a position marker. If this position marker appears in the upper left hand corner of the screen, then that portion of the image represents the dorsal aspect of the image slice (Figure 96). Although the

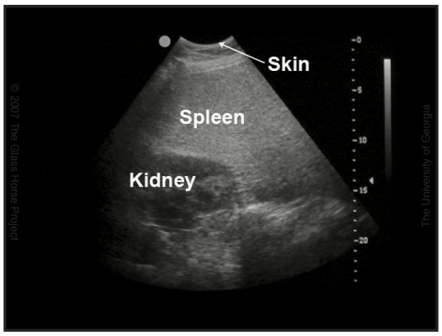

Figure 92. Ultrasound image of a horse's spleen and left kidney

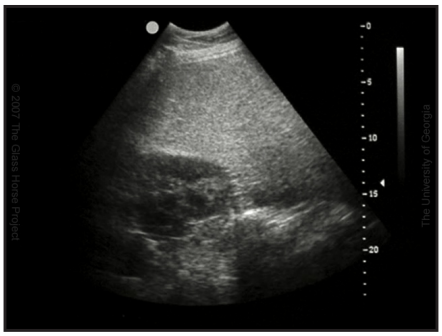

Figure 93. Tissues near the transducer are at the top of the image.

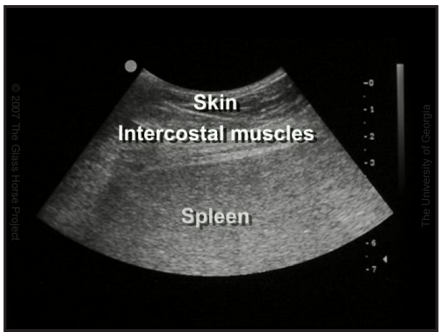

Figure 94. Ultrasound image showing skin, intercostal muscles, and spleen

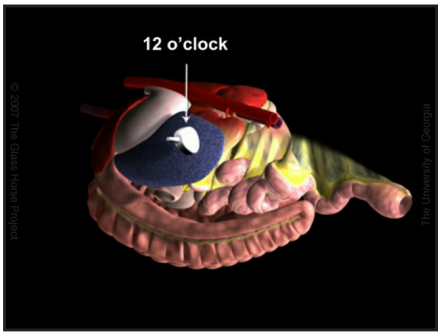

Figure 95. Common position of the ultrasound transducer

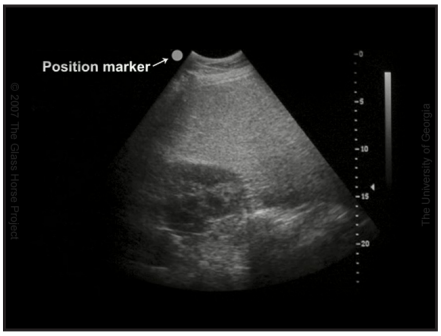

Figure 96. Position marker on the dorsal aspect of the image

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transducer works perfectly well if it is flipped 180 degrees on the patient, with the transducer marker at the 6 o'clock position, the machine will project the image slice with the ventral aspect of the image on the left hand side of the screen. Most machines can flip the displayed image left or right and/or up or down at the press of a button, without moving the transducer.

The concept of transducer orientation on the patient relative to what is displayed on the screen is important when the transducer is rotated to view the same tissue or organ using different planes of projection. As an example, a **short axis or transverse view** through the kidney is obtained when the transducer marker is positioned at 12 o'clock on the patient, over the spleen and kidney at the 15th intercostal space (Figure 97). By rotating the transducer 90 degrees, a **long axis or sagittal view** of the left kidney is generated (Figure 98). If the transducer marker is rotated back to the 12 o'clock position while the transducer is moved dorsally in the intercostal space, the head or dorsal edge of the spleen can be seen on the image (Figure 99). This portion of the spleen was not present on either of the other two views. With the transducer in the transverse plane, the screen marker is on the left, and the dorsal edge of the spleen is on the left side of the displayed image.

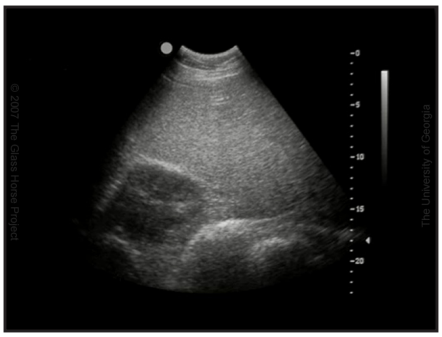

Figure 97. Transverse ultrasound view through the kidney

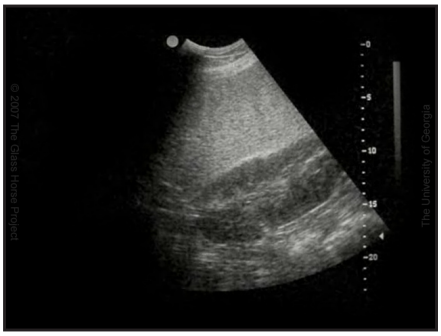

Figure 98. Sagittal ultrasound view through the kidney

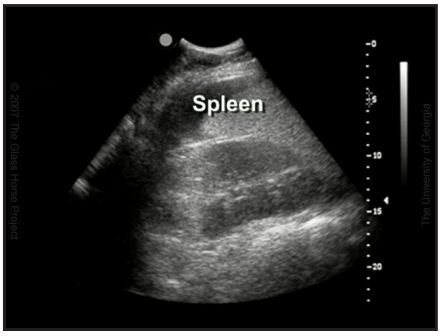

Figure 99. Ultrasound view of the kidney and dorsal edge of the spleen

How the Image Is Generated

It is obvious to anyone who has done any ultrasonography that the images are generated instantaneously. In an effort to make the displayed image make more sense to the novice, in this section we are going to use a bit of artistic license. As ultrasound waves project through the body, they are reflected by tissue interfaces and sensed as "**echoes**" (Figure 100). If adjacent tissues have the same acoustic impedance, no sound is reflected, and sound waves penetrate into the deeper tissues. While denser tissues have greater acoustic impedance, it is the interface between adjacent tissues or between tissues within the same organ that determines how much of the sound wave is reflected back to the transducer. The more sound reflected to the trans-

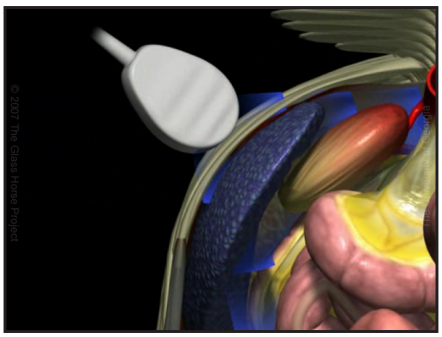

Figure 100. Reflection of ultrasound waves by tissue interfaces

ducer, the whiter the image appears on the screen. These tissue interfaces are called **echogenic** or **hyperechoic** (Figure 101). In contrast, less dense tissues reflect less sound and are called **anechoic** or **hypoechoic** and appear blacker (Figure 102).

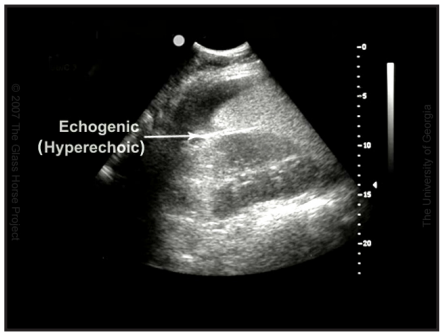

Figure 101. Echogenic or hyperechoic aspects of an image

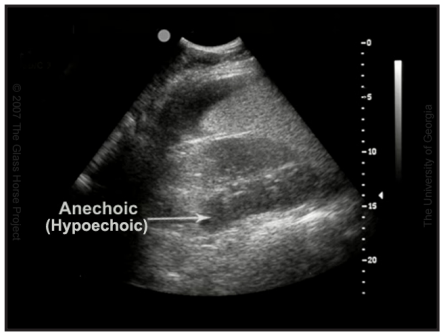

Figure 102. Anechoic or hypoechoic aspects of an image

In this ultrasound image of the horse's spleen and kidney, the whiter portions of the image close to the transducer represent the skin, fascia, intercostal muscles, and peritoneum. Just deep to these lines is a fairly thick whitish, and therefore uniformly dense, tissue. This is the **spleen**, which is positioned against the left body wall (Figure 103). A quick reference to the scale shows the thickness of the spleen to be about 10 to 14 cm in this area. The dorsal aspect of the image is to the left, so the most dorsal aspect of the head of the spleen is off the screen to the left. Slightly deeper into the image are **parallel bright white lines** a short distance away from and parallel to the medial edge of the spleen. These are the **fibrous capsules** on the medial side of the spleen and the lateral side of the left kidney. Based on anatomic specimens, the caudal pole of the left kidney should be 6 to 8 cm thick and approximately 15 cm in height. The renal pelvis contains fairly dense fibrous tissue, and the terminal recesses contain less tissue than the surrounding medulla. Therefore, on the ultrasound image, the renal pelvis appears as a white line near the center of the kidney, the less dense renal cortex and medulla appear grayer, and the terminal recesses, which are the least dense areas near the pelvis, appear darkest. A quick comparison of the ultrasound image with a kidney sliced along the same plane helps make these points even more obvious.

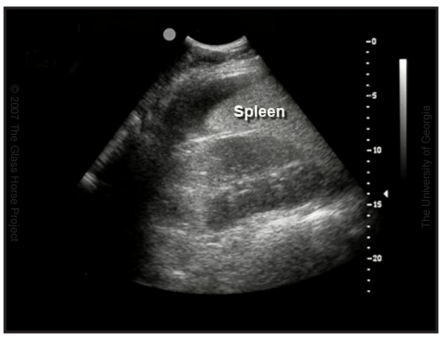

Figure 103. Ultrasound image of the spleen against the left body wall

Ultrasonography can be used to identify **gas, fluid, and interfaces between gas and fluid** within the intestine. Examples of these are best depicted by scanning the horse's stomach; this image was obtained via the 12th intercostal space. To produce this image, the ultrasound beam passed through the spleen, which is adjacent to the left abdominal wall, and intersected the wall of the greater curvature of the stomach (Figure 104). The large **splenic vein** can be used as a landmark as it is near the wall of the stomach. With the orientation marker positioned dorsally, this ultrasound machine places the dorsal aspect of the image on the left side of the screen. Given that it is primarily the **density differences at tissue interfaces** that are responsible for reflecting sound back to the transducer, more sound waves should echo

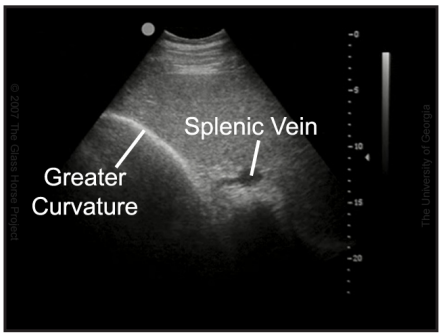

Figure 104. Ultrasound image of the greater curvature of the stomach and the splenic vein

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back to the transducer if two adjacent interfaces have markedly different acoustic impedances. Interfaces between soft tissues and gas are excellent examples of this concept. The soft tissue of the stomach wall has an acoustic impedance that is several thousand fold greater than that of the free gas inside the lumen of the stomach. Consequently, the image at this soft tissue to gas interface appears as a fuzzy **hyperechoic border** (Figure 105). Since most of the sound waves at this interface are reflected, and the free gas in the lumen has extremely low impedence, the rest of the lumen of the stomach appears darker as sound is neither penetrating nor reflecting from the lumen. This concept is even easier to see in an image taken from a horse with edema in the wall of the stomach. In this image, the **edematous fluid** in the stomach wall is **hypoechoic**, and it offsets the **hyperechoic serosal surface** of the stomach from the hyperechoic interface between the mucosal surface and the gas in the lumen (Figure 106).

With this background material in mind, you are now ready to interpret ultrasound images of normal horses and horses with gastrointestinal diseases. As you examine these images, take note of the following: the orientation of the transducer; the orientation of the image on the screen; the depth of penetration of the beam; and the locations of gas, fluid, and tissue interfaces.

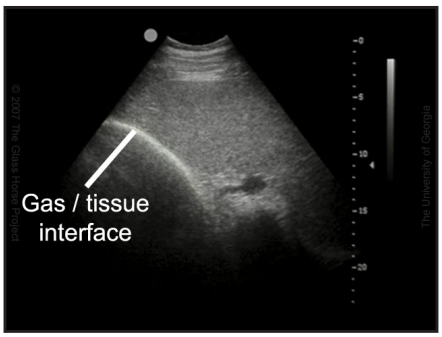

Figure 105. Ultrasound image of a soft tissue to gas interface

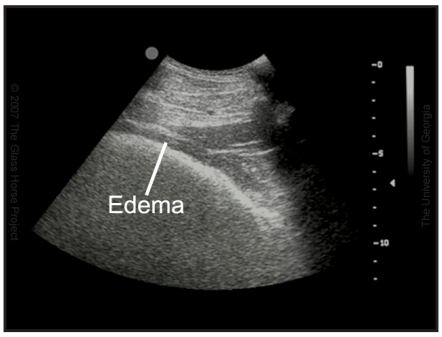

Figure 106. Ultrasound image of edema in the stomach wall

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Diseases

Stomach

Ulcers (Ulceration)

Gastric ulcers are associated with a wide variety of clinical signs and degrees of abdominal pain in horses, and, in fact, as many as 50% of asymptomatic foals and 80% of racehorses have gastric ulcers. Gastric ulcers usually are identified in the **squamous mucosa** near the **margo plicatus** (Figure 107), requiring that gastroscopy be performed before a diagnosis can be made. Clinical signs of gastric ulcers tend to be those associated with **low-grade pain**, such as poor appetite, weight loss, stretching, and pawing. Affected foals commonly roll onto their backs and remain in that position; they tend to present with bruxism, salivation, lip curling, and low-volume diarrhea. Occasionally, pyloric or duodenal ulcers in foals cause stricture formation and obstruction of gastric outflow. Treatment of affected horses involves the administration of drugs that **reduce gastric acidity** by inhibiting gastric acid secretion. Except for foals with pyloric outflow obstruction secondary to duodenal ulcers, the prognosis is generally good.

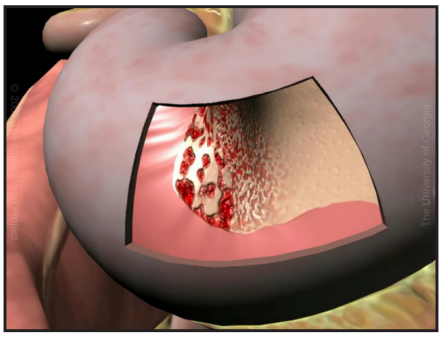

Figure 107. Ulceration of the gastric mucosa near the margo plicatus

Gastric Dilation (Distention)

Dilation of the stomach usually occurs secondary to conditions affecting the small intestine, with the most common causes being the buildup of fluid from the small intestine because of ileus, obstruction of the small intestinal lumen, strangulation obstruction involving the small intestine, and severe inflammation of the small intestine. Occasionally, fluid accumulates in the stomach in horses with large colon displacements, presumably due to impingement on the duodenum as it crosses dorsal to the base of the cecum. **Gastric dilation** also can occur after ingestion of highly fermentable plant material (e.g., grass clippings, excess corn, or grain). Ingestion of this material results in the generation of an excessive amount of gas, closure of the gastroesophageal junction, and distention of the stomach (Figure 108). As a result, affected horses become acutely painful and have increased heart and respiratory rates due primarily to pain and diaphragmatic pressure. If the dilation is secondary to a problem involving the small intestine, the animal may appear toxic, the peritoneal fluid may be abnormal (due to increased white blood cell count and protein concentration), and distended small intestine may be palpable on rectal exam. Horses with gastric dilation are at risk of gastric rupture, which occurs most often along the greater curvature.

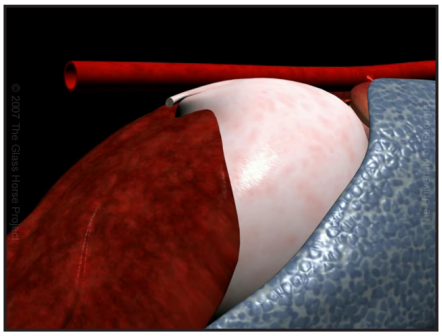

Figure 108. Distention of the stomach

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Treatment involves decreasing gas/fluid pressure with a stomach tube and then determining whether or not the condition is primary or secondary. If the dilation is primary, the horse will no longer be painful after relief of excessive gastric pressure. If, however, the dilation is secondary to a small intestinal problem, the relief will be transient. If gastric rupture occurs, the horse will abruptly stop showing signs of pain and will deteriorate rapidly. Ingesta will be evident in the peritoneal fluid, and euthanasia is indicated. The prognosis depends on the underlying cause.

Gastric Impaction (Obstruction)

Impaction of the stomach is a **rare cause of colic** in horses and usually is not recognized before surgery. Impaction of the stomach with dry or poorly masticated feed stretches the wall of the stomach and **may displace the spleen caudally** (Figure 109). It is important to note, however, that caudal displacement of the spleen is not pathognomonic for gastric impaction. Affected horses may show signs of moderate to severe pain, and most will not be ill unless the initiating cause is grain overload. Horses with impacted ingesta in the stomach may be violently painful and require **exploratory surgery**. Treatment generally requires surgery, during which the impaction may be relieved by intragastric administration of water through a needle inserted through the stomach wall. Postoperative care may include gavage of the stomach contents by instilling water into the stomach and draining it out through a stomach tube. Because diagnosis is often delayed and difficulties are frequently encountered in relieving the impaction, the prognosis is guarded to poor.

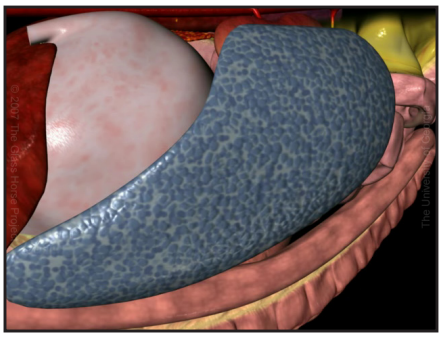

Figure 109. Gastric impaction

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Small Intestine

Spasmodic Colic (Spasm)

Spasmodic colic may be the most commonly diagnosed and least understood form of colic in horses. In the majority of cases, the cause of spasmodic colic is not determined, but it is presumed that it occurs due to **spasm** or **cramping of intestinal musculature** (Figure 110). Some veterinarians suggest that it is initiated by mild buildup of gas in the lumen of the intestine, whereas others suggest that it is a response to undigested feed, ingestion of fresh grass, alterations in the nerve supply to the intestine, or increased stress. As such, the diagnosis is based on the **lack of other findings** and the fact that the abdominal pain is relieved by administration of mild analgesics or spasmolytic agents. The severity of abdominal pain in most horses diagnosed with spasmodic colic is mild, and the signs occur intermittently. Some veterinarians report that it is possible to hear rumbling intestinal sounds without the aid of a stethoscope.

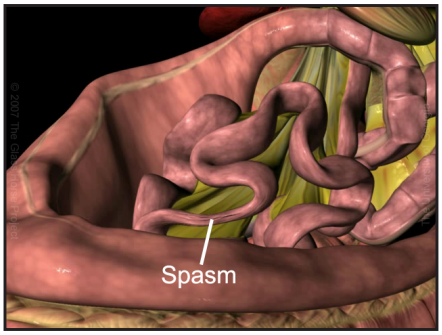

Figure 110. Spasm of the intestinal musculature

Small Intestinal Strangulation through Mesenteric Rent (Strangulation Obstruction)

Small intestinal strangulation commonly involves a defect in the small intestinal mesentery called a **mesenteric rent**. Problems arise when normal peristaltic contractions cause a loop of the jejunum to pass through a mesenteric rent (Figure 111). As the **thicker-walled ileum** is drawn into the opening, the outflow of blood and lymph from the intestinal loop is impeded, the intestinal wall becomes edematous, and the intestinal lumen becomes occluded (Figure 112). Continued inflow of arterial blood in the presence of an obstruction to venous outflow results in the rupturing of capillaries in the mesentery and intestinal wall (Figure 113). Eventually, the affected intestine becomes ischemic, and intestine proximal to the **strangulation** distends with gas and fluid.

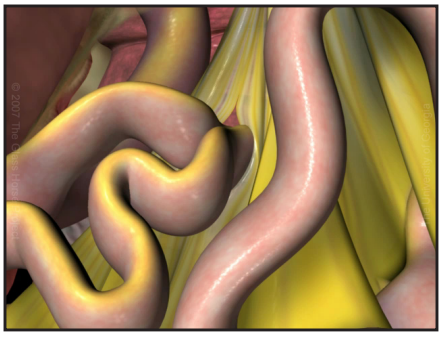

Figure 111. Movement of jejunum through a mesenteric rent

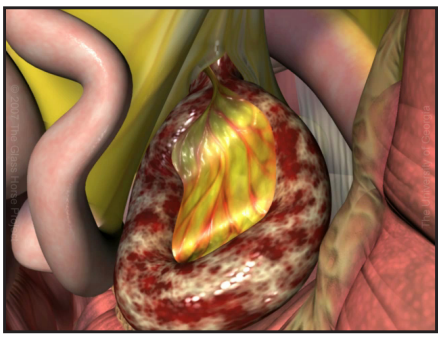

Figure 112. Strangulation of the jejunum and ileum

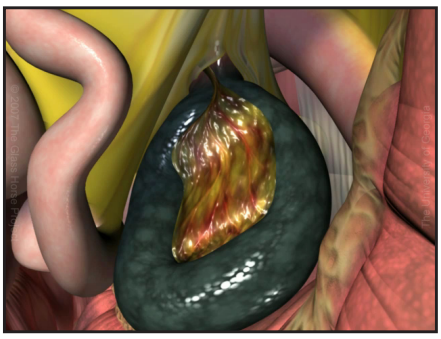

Figure 113. Intestinal ischemia as a result of strangulation obstruction

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Affected horses are painful, toxemic, hemoconcentrated, and dehydrated, and they have **distended loops of small intestine** on rectal palpation (Figure 114). Treatment involves surgery under general anesthesia to remove the devitalized intestine and perform a resection and anastomosis. The **prognosis** for survival is **fair** but can be improved if the condition is recognized and surgery performed early in the course of the disease. The most common complication associated with this condition is recurrent colic due to the development of postoperative adhesions.

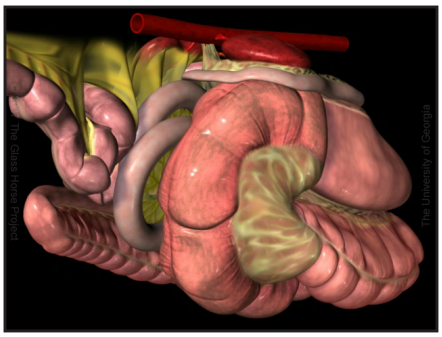

Figure 114. Distended loops of jejunum may be palpated on rectal examination.

Adhesions (Obstruction)

Adhesions involving the jejunum usually develop as a complication of previous **small intestinal surgery** or because of parasite migration, abdominal abscesses, penetrating abdominal wounds, or serosal inflammation (e.g., **proximal enteritis**). Initially, the adhesions are comprised of fibrinous tags between loops of bowel or between the bowel and mesentery (Figure 115). Under normal circumstances, the **fibrinous adhesions** are removed by the body's fibrinolytic system (Figure 116). If this system is ineffective, usually due to excessive intra-abdominal inflammation, the fibrinous adhesions are invaded by fibroblasts, and the adhesions become **fibrous** (Figure 117). As these adhesions mature, they produce kinks in the intestine, which can result in **intestinal obstruction** (Figure 118). Alternatively, the adhesions may create spaces through which intestine can become strangulated.

Affected horses often have a history of a gradual onset of colic and weight loss, and in many instances the pain occurs after the horse eats. In some horses,

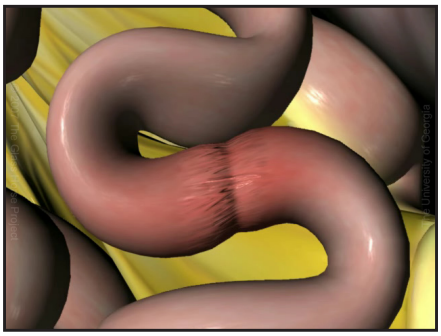

Figure 115. Fibrinous adhesions between adjacent loops of bowel

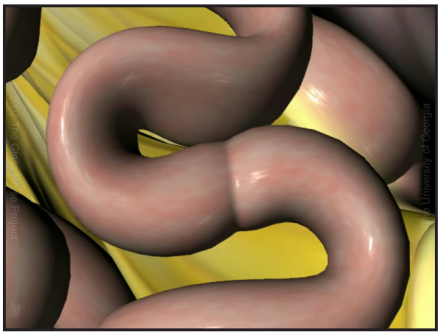

Figure 116. The fibrinous adhesions have been removed by fibrinolysis.

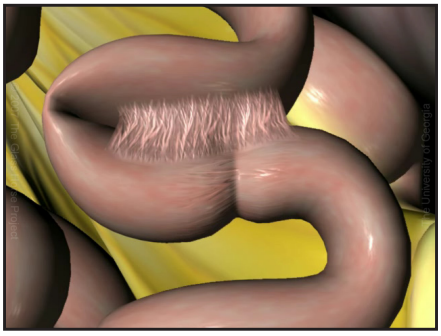

Figure 117. Permanent fibrous adhesions

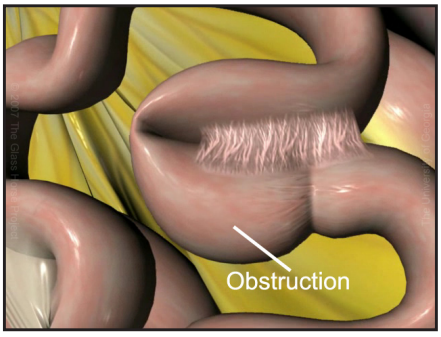

Figure 118. Obstruction of the bowel due to fibrous adhesions

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slightly distended intestine can be felt on rectal exam. In a recent study of small intestinal surgery in horses, adhesions caused problems in approximately 20% of the horses after surgery, and signs of colic developed most often within the **first 60 days after surgery**. Treatment of adhesions may involve changing the horse's diet to facilitate movement of ingesta, or, more often, surgery to remove the affected segments of intestine. In an effort to prevent the formation of postoperative adhesions, several treatments have been evaluated during surgery. The most commonly used measure involves intra-abdominal instillation of sterile carboxymethylcellulose at the end of surgery to coat intestinal serosal surfaces. The prognosis is poor if the adhesions cause clinical problems within the first 60 days after surgery, but it improves if they become evident later.

Epiploic Foramen Entrapment (Strangulation Obstruction)

The epiploic foramen, which is bounded by the visceral surface of the caudate lobe of the liver, the portal vein, the caudal vena cava, and the pancreas, opens into the omental bursa (Figure 119). In the majority of horses with epiploic foramen entrapment, the **distal jejunum and ileum** move from the left side of the abdomen and through the foramen, tearing the omentum in the process (Figure 120). As the entrapped intestine becomes edematous and thickened, it also becomes strangulated, discolored, and devitalized (Figure 121). Jejunum proximal to the strangulation distends with gas and fluid (Figure 122).

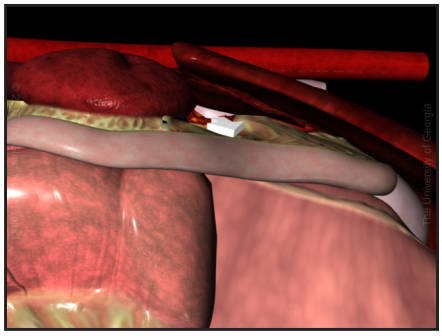

Figure 119. Opening into the epiploic foramen

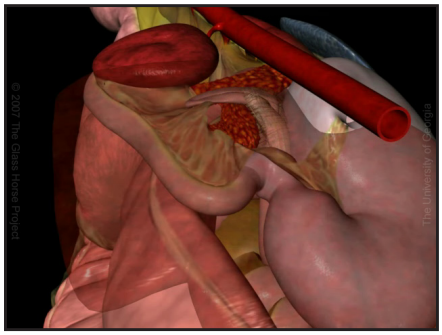

Figure 120. Movement of the small intestine into the epiploic foramen

Most often, affected horses are painful, although some horses may appear depressed. Although epiploic foramen entrapments tend to occur in horses older than 8 years of age, there appears to be no correlation between age and the size of the epiploic foramen. Surgery must be performed to remove the entrapped intestine and, if the intestine is devitalized, to perform a resection and anastomosis. Very often these horses require intensive postoperative care, and the most commonly encountered complications are ileus and adhesions. The prognosis for survival is fair to good, depending on the duration of the condition preoperatively.

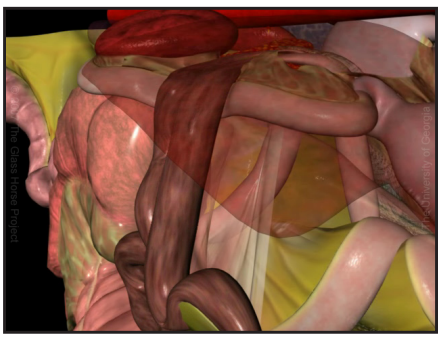

Figure 121. Ischemia of the strangulated jejunum and ileum

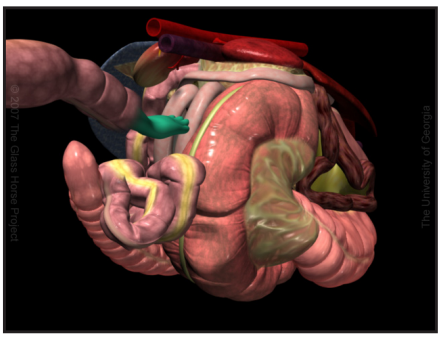

Figure 122. Distended loops of jejunum may be palpated on rectal exam

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Pedunculated Lipoma (Strangulation Obstruction)

Pedunculated lipomas are fatty tumors that may cause small intestinal strangulation in horses more than **12 years of age**; they appear to occur more commonly in geldings. The lipoma forms on the end of a pedicle of tissue originating from the mesentery (Figure 123). The pedicle wraps around a loop of jejunum and its mesentery, causing obstruction of the intestinal lumen (Figure 124). Due to the weight of the lipoma and the development of edema in the affected loop of intestine, the knot tightens and strangulates the intestine (Figure 125). Small intestine proximal to the strangulation then distends with gas and fluid (Figure 126) and can be felt during the rectal examination. While one might expect affected horses to exhibit signs of severe abdominal pain, very often horses with pedunculated lipomas appear **depressed**. Surgical intervention is required to resect the lipoma and remove any devitalized tissue. The prognosis is good, with the most commonly encountered complications being ileus and intra-abdominal adhesions.

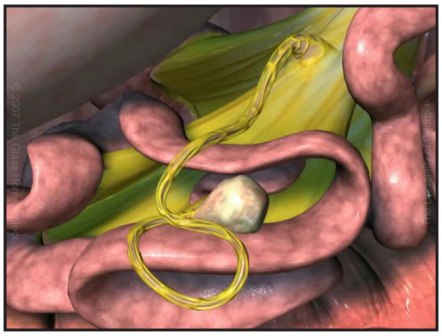

Figure 123. A pedunculated lipoma originating from the mesentery

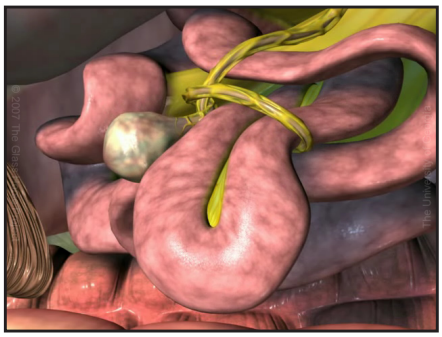

Figure 124. Entrapment of a loop of intestine by the pedunculated lipoma

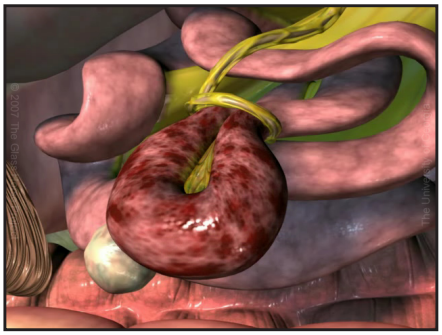

Figure 125. Strangulation obstruction of a section of intestine

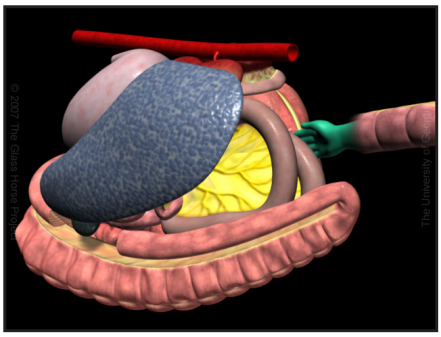

Figure 126. Distended loops of intestine may be palpated on rectal exam.

Inguinal Hernia of Small Intestine (Strangulation Obstruction)

To understand what happens during an inguinal hernia, it is important to be familiar with the relevant anatomy. Within the scrotal sac and inguinal canal in male horses is a strong tissue called the vaginal tunic. Deep to the vaginal tunic are the testis, testicular blood vessels, epididymis, and

ductus deferens (Figure 127). The testicular vessels and ductus deferens pass through the vaginal ring, an evagination of the parietal peritoneum that forms the communication between the cavity of the vaginal tunic and the peritoneal cavity. Inguinal hernias occur in stallions and most often involve a relatively short segment of distal jejunum and ileum (Figure 128). The condition develops when the small intestinal segment passes through the vaginal ring, and it often occurs **after the stallion has bred a mare, fallen, or undergone strenuous exercise**. After becoming edematous and

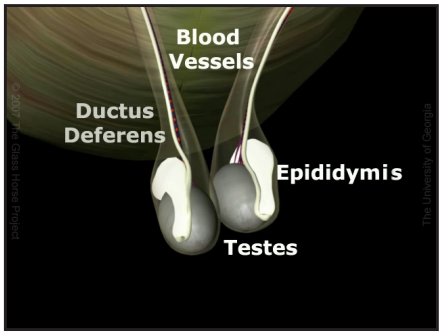

Figure 127. The normal contents of the inguinal canal

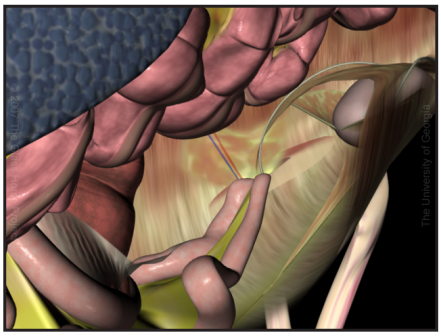

Figure 128. Movement of jejunum and ileum through the vaginal ring

thickened, the affected intestine then becomes ischemic (Figure 129). Due to the excessive pressure within the inguinal canal, the testicle on the affected side also may become ischemic. Jejunum immediately proximal to the strangulated segment distends with gas and fluid and is fixed in place in the inguinal region (Figure 130).

Most affected stallions exhibit clinical signs of acute abdominal pain, although some, particularly those of the Tennessee Walking Horse breed, may be more stoic. In any case, the testicle on the affected side becomes enlarged, swollen, cold, and painful, and intestine is palpable entering the inguinal ring on rectal exam. Surgery is performed to remove entrapped intestine and, if the intestine is devitalized, to perform a resection and anastomosis. This will require both a ventral midline and an inguinal approach. The prognosis for survival is fair, with the most commonly encountered complications being ileus and intra-abdominal adhesions.

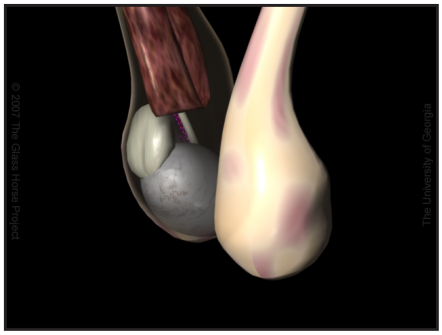

Figure 129. Strangulation of the jejunum and ileum

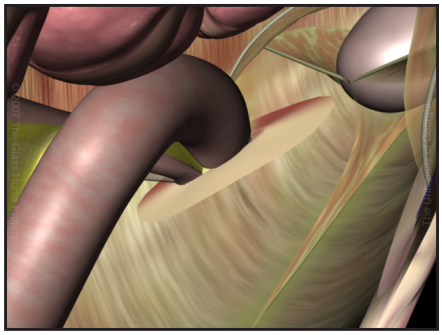

Figure 130. Distention of jejunum proximal to the strangulated intestine

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Ileal Impaction (Obstruction)

Ileal impaction is a common cause of small intestinal obstruction in specific regions of the world. Normally, peristaltic waves in the small intestine force the ingesta through the ileocecal orifice and into the cecum, where nutrients and water are absorbed. An impaction develops when the ingesta cannot pass through the orifice; the impaction enlarges as additional ingesta moves from the jejunum to the ileum (Figure 131). The fluid that normally is reabsorbed in the cecum accumulates in the lumen of the jejunum proximal to the impaction, and the intestine distends (Figure 132). The increase in intraluminal pressure causes additional fluid to be secreted into the intestinal lumen, which further increases the severity of intestinal distention.

Affected horses are on average about 8 years of age, and the condition occurs most commonly in the **southeastern part of the United States** and in certain parts of the European continent. Although the condition has been associated with changes in feed, with dry, finely ground feed, and with feed having a high fiber content (**coastal Bermuda hay**), a cause-and-effect relationship has not been proven. There has also been an association between this condition and irritation at the ileocecal junction resulting from tapeworm infestation. Affected horses have an acute onset of pain that is mild to moderate in nature. Approximately half of the affected horses will have gastric reflux, whereas almost all will have reduced intestinal sounds and distended intestine on rectal exam. Early on it is possible to feel the impacted ileum, which is approximately 6 cm in diameter, extending from right of midline at the cecal base obliquely downward to the left ventral abdomen (Figure 133). After 8 to 10 hours, however, distention of the jejunum obscures the impaction, making the diagnosis more difficult to make (Figure 134). At this stage of the disease, nasogastric reflux occurs.

If the impaction is identified before the jejunum distends, affected horses may be treated with mineral oil, analgesics, and intravenous fluids. Once the jejunum has distended and there is reflux of small intestinal secretions into the stomach, surgery is required to break down the impaction and massage it into the cecum. Many surgeons inject carboxymethylcellulose into the lumen of the ileum, mix it with the impacted material, and then move everything into the cecum. The prognosis is good, with survival rates exceeding 75% in many clinics.

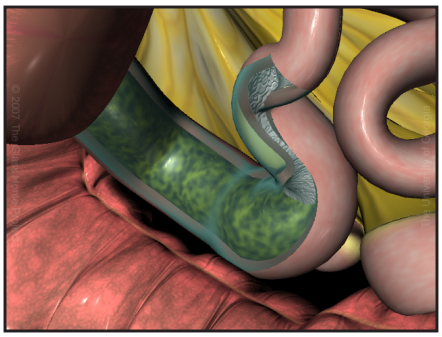

Figure 131. Impaction of the ileum with ingesta

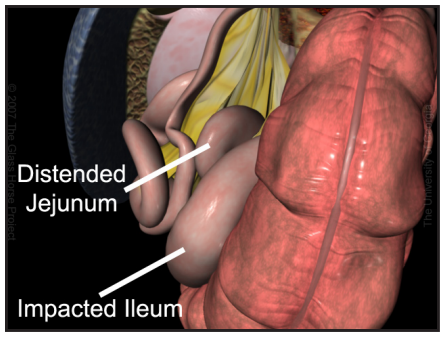

Figure 132. Jejunum proximal to the impaction begins to distend.

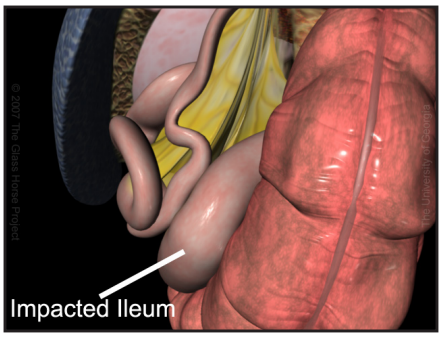

Figure 133. The impacted ileum can be palpated early in the disease.

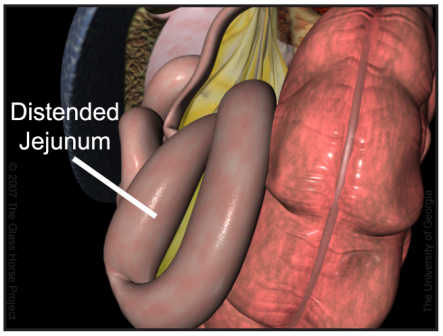

Figure 134. Gas-distended loops of jejunum make it difficult to find the impaction.

Small Intestinal Volvulus (Strangulation Obstruction)

Small intestinal volvulus can affect all or a portion of the small intestine. It is theorized that a volvulus develops when peristalsis in a normal segment of intestine causes it to move around a **distended, immotile segment** of intestine (Figure 135). This causes a twisting of the mesentery. Once the mesentery rotates 360 degrees, the mesenteric vessels are occluded, causing the affected intestine to become ischemic (Figure 136). The rotation also obstructs the intestinal lumen. If the entire small intestine is affected, it becomes distended, thickened, and eventually, necrotic (Figure 137).

Affected horses with volvulus show the classical signs of small intestinal strangulation obstruction (abdominal pain, reflux, poor tissue perfusion, reduced intestinal sounds, and distended small intestine on rectal exam; Figure 138). The peritoneal fluid will change in color, cell count, and protein content. **Metabolic acidosis** is common. Surgical intervention is required to reduce the volvulus, and, if necessary, resect any devitalized intestine. As a general rule, resection of more than 50% of the total length of small intestine is associated with a poor prognosis. Overall, however, the prognosis is fair to good, depending on the duration of the volvulus prior to surgery.

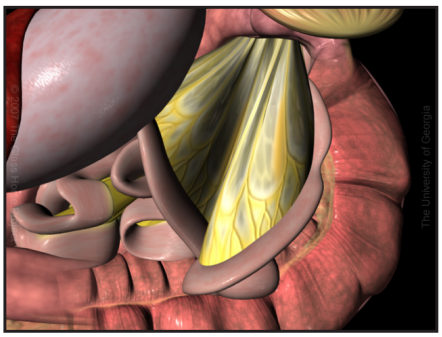

Figure 135. Movement of normal intestine past an immotile segment

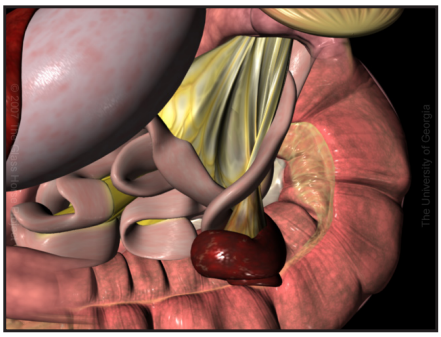

Figure 136. Ischemia of the intestine due to occlusion of mesenteric vessels

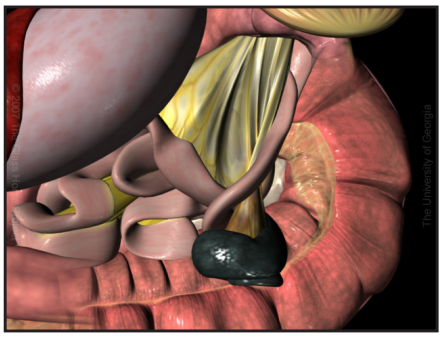

Figure 137. Strangulation obstruction of the affected intestine

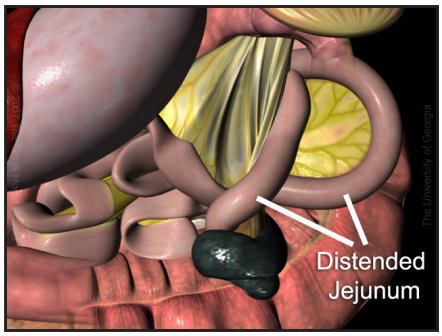

Figure 138. Distended loops of intestine may be palpated on rectal examination.

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Ileocecal Intussusception (Strangulation Obstruction)

Ileocecal intussusception is a condition that has been associated with mucosal irritation due to **tapeworm infection**. This condition occurs when a peristaltic wave causes the ileum to invaginate through the ileocecal orifice (Figure 139). Additional muscular contractions then cause the ileum and, in many cases, several feet of the jejunum to move into the cecum (Figure 140). The invaginated small intestine, which is called the **intussusceptiens**, becomes strangulated, causing it to thicken and enlarge within the lumen of the cecum (Figure 141). Jejunum proximal to the intussusception then distends with gas and fluid (Figure 142).

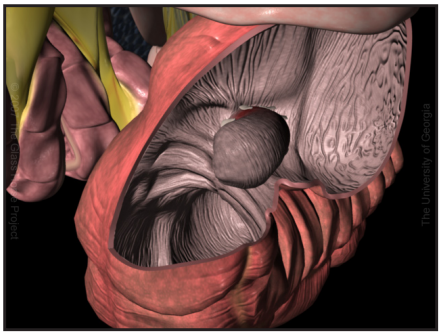

Figure 139. Invagination of the ileum through the ileocecal orifice

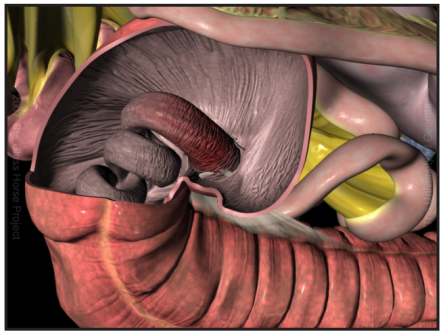

Figure 140. Ileum and jejunum within the lumen of the cecum

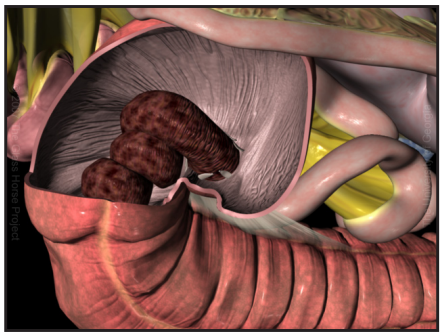

Figure 141. Strangulation obstruction of the affected intestine

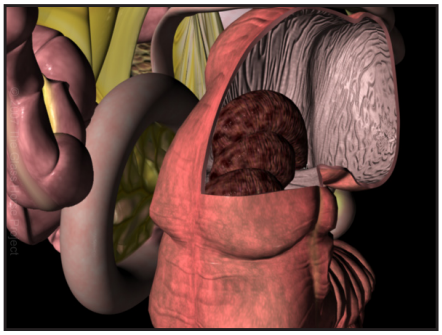

Figure 142. Distended loops of intestine may be palpated on rectal exam.

Intussusceptions sometimes occur after deworming and abrupt dietary changes and with ascarid or tapeworm infestations; they occur most commonly in horses less than 3 years of age. Two clinical syndromes have been described for horses with ileocecal intussusception. In one scenario, the intussusception occurs acutely, causing the horse to be severely painful and to have reduced gastrointestinal sounds, gastric reflux, and distended loops of intestine on rectal palpation. When the more chronic form of the disease develops, affected horses are in poor physical condition, are mildly or moderately painful, especially after eating, and may have a low-grade fever. Peritoneal fluid from these horses may be normal. In about 30% of affected horses, a tight tubular mass (the ileum and part of the jejunum within the cecum) may be palpated in the upper right quadrant during the

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rectal examination. Intussusceptions, particularly those involving the small intestine, also may be identified by transabdominal ultrasonography.

Treatment involves surgical intervention to reduce the intussusception, if possible, and to remove the affected intestine and perform an anastomosis. The prognosis is guarded for horses with acute ileocecal intussusception as necrosis of the ileal stump may result in septic peritonitis, abscess, and adhesions. The prognosis is better for horses with chronic intussusception, but the postoperative recovery may be slow due to chronic distention and thickening of the jejunum.

Proximal Enteritis (Enteritis)

Proximal enteritis is an inflammatory condition that affects the **duodenum and proximal half of the jejunum**. Initially, the duodenum becomes inflamed and then distends with fluid and gas (Figure 143). As the disease progresses, distended loops of jejunum are identified in the middle of the abdomen (Figure 144). Because there is no physical blockage of the jejunum, it is not as tightly distended as with a small intestinal strangulation (Figure 145). The disease syndrome is characterized by ileus, nasogastric reflux, and an increased risk for development of laminitis. The cause is unknown, though some clinicians have suspected that clostridial or salmonella organisms may be involved. There appears to be a higher prevalence of the disease in horses eating **grain or pelleted diets**.

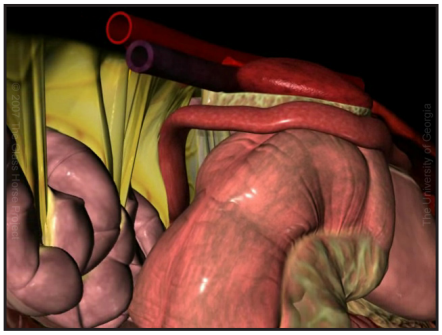

Figure 143. Inflammation and distention of the duodenum

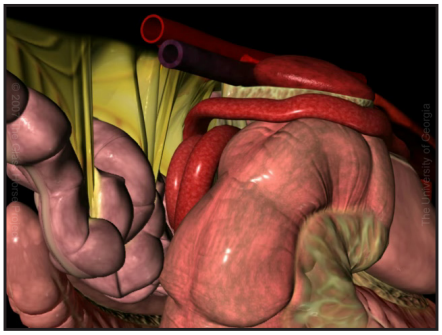

Figure 144. Distended loops of jejunum in the middle of the abdomen

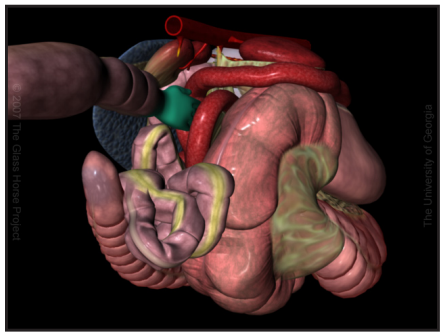

Figure 145. Moderately distended loops of jejunum may be palpated.

Although affected horses initially exhibit signs of acute abdominal pain, these are replaced with clinical signs of **depression**, dehydration, hemoconcentration, and, in some cases, a low-grade fever. Classically, there is voluminous gastric reflux, which in some cases is orange in color, and moderate distention of small intestine on rectal exam. Peritoneal fluid may be sanguinous and generally has increased protein content and a normal to mildly increased white blood cell count. Transabdominal ultrasound examination reveals fluid-distended loops of small intestine with thickened, corrugated walls. Due to the clinical signs and laboratory findings, it can be very difficult to distinguish horses with this disease from those with ileal impaction or small intestinal strangulation.

The mainstays of treatment are removal of gastric reflux and intravenous administration of balanced electrolyte solutions; some clinicians administer lidocaine as a continuous intravenous infusion, and others administer serum or drugs designed to combat the ill effects of endotoxemia. Affected horses are maintained without feed until the reflux subsides, at which time hay is gradually reintroduced. The prognosis for survival is generally good, although laminitis and thrombophlebitis are serious complications that may arise.

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Cecum

Cecal Tympany (Distention)

Gas distention of the cecum, known as **cecal tympany**, occurs commonly in horses with colonic displacements, colon volvulus, or obstruction of the small colon. Less often, cecal tympany occurs as a primary disease, presumably due to the rapid fermentation of lush pasture grasses or an abrupt change in diet. The most obvious clinical findings in affected horses are distention of the abdomen, tight paralumbar fossae, pain, tachycardia, and tachypnea. Auscultation and percussion of the abdomen over the right paralumbar region reveal a high-pitched pinging sound characteristic of a distended viscus, and the rectal examination reveals a **taut ventral cecal band**, coursing diagonally from the right dorsal to the left ventral abdomen (Figure 146). In fact, the cecum may be so distended that it prevents palpation of other portions of the gastrointestinal tract.

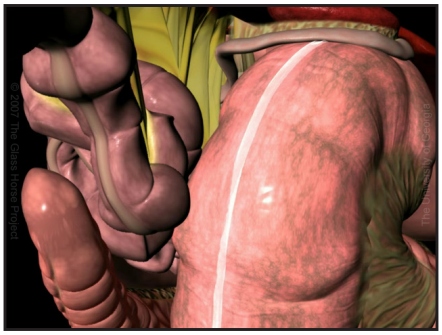

Figure 146. Gas distention of the cecum

Treatment of primary cecal tympany includes removal of the gas through a trocar placed aseptically through the right paralumbar fossa. If cecal tympany is secondary to another condition, correction of the underlying problem will resolve the tympany. The prognosis for survival from primary cecal tympany is good; the prognosis for horses with secondary cecal tympany is also good but ultimately depends on the severity of the underlying condition.

Cecal Impaction (Obstruction)

Normally, hydrated ingesta enters the cecum from the ileum. In a well-coordinated series of muscular contractions, the ingesta is moved from the cecal base to the apex and finally through the cecocolic orifice into the right ventral colon (Figure 147). However, in horses that develop cecal impactions, the coordinated pattern of cecal muscular activity is disrupted, and the **ingesta accumulates in the base of the cecum** (Figure 148). With time,

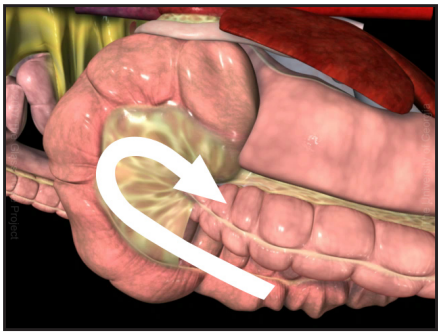

Figure 147. Normal motility pattern of the cecum

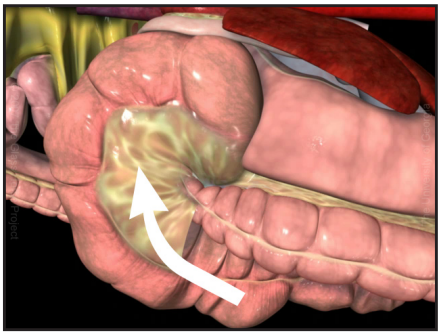

Figure 148. Cecal motility is altered in horses with cecal impaction.

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fluid is reabsorbed, and the ingesta becomes dehydrated, forming an impaction (Figure 149). As the impaction persists, additional ingesta accumulates in the cecal body and apex. The weight of the impacted material makes the **ventral cecal band** easy to identify during the rectal examination (Figure 150). In contrast, the large colon may be difficult to locate because its motility patterns remain normal, causing it to continue to empty. Cecal impaction has been associated with ingestion of coarse feed, with poor teeth, and with insufficient water supply or reduced intake of water. It has also been linked to hospitalization, general anesthesia, and other diseases requiring prolonged treatment with nonsteroidal anti-inflammatory drugs.

Most affected horses intermittently exhibit clinical signs of mild abdominal pain and are not toxemic. Cecal impactions tend to occur in horses about 8 years of age and are more often associated with abrupt rupture than are impactions involving any other part of the intestinal tract. Treatment requires intravenous and oral administration of fluids, mild analgesics, and laxatives. **Spontaneous rupture of the cecum** has been reported in almost 50% of cases; the client should be aware of the possibility of abrupt cecal rupture, and medical therapy should be aggressive. Surgical intervention may be necessary, in which case a jejunocolic anastomosis may be performed to bypass the cecum. Due to the possibility that the cecum may rupture spontaneously, the prognosis is guarded.

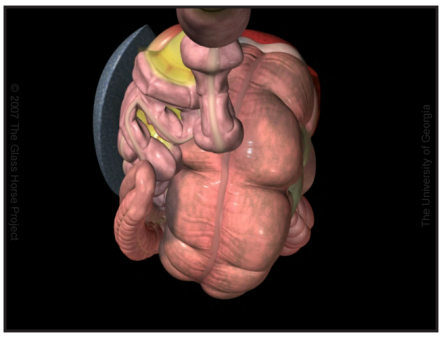

Figure 149. Impaction of the base of the cecum with dry ingesta

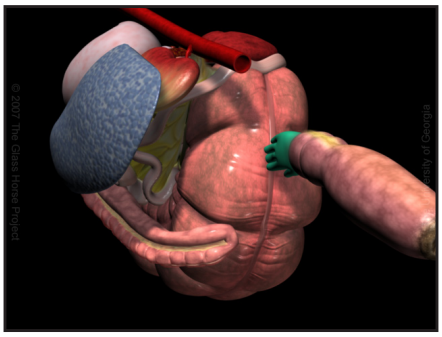

Figure 150. Identification of the ventral cecal band during rectal examination

Cecocolic Intussusception (Strangulation Obstruction)

Cecocolic intussusception is an **uncommon cause of colic** in horses, accounting for less than 1% of horses referred to university clinics with abdominal pain. This condition involves the telescoping of the apex of the cecum into the body (Figure 151) followed by the invagination of the cecum through the cecocolic orifice and into the lumen of the right ventral colon (Figure 152). Although the underlying cause of this condition is not known, it is most commonly associated with infection with the equine tapeworm,

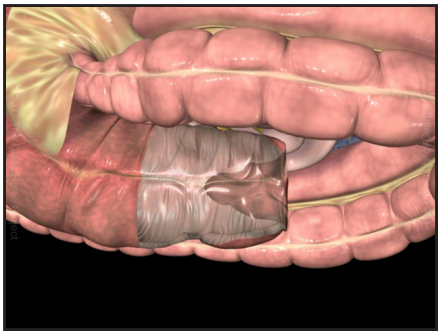

Figure 151. Telescoping of the apex of the cecum into the body

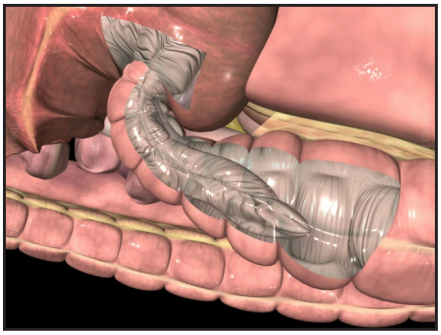

Figure 152. Invagination of the cecum into the lumen of the colon

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Anaplocephala perfoliata. The reason for this association is the presence of inflammatory lesions, which occur at the site of attachment of the tapeworms in the cecum. Other possible causes of cecocolic intussusception include the administration of organophosphates and parasympathomimetic drugs.

Young horses, typically **less that 3 years of age**, appear to be at greatest risk for developing these conditions. Clinical signs associated with cecocolic intussusception are variable; some affected horses have chronic, intermittent episodes of mild-to-moderate abdominal pain accompanied by weight loss, scant feces, and an intermittent fever, whereas other horses have an acute onset of severe, unrelenting abdominal pain. It is presumed that the more chronic form of the disease is seen in horses in which only part of the cecum has passed into the cecocolic orifice, whereas acute pain occurs in horses in which the cecocolic orifice is obstructed by the cecum, resulting in distention of the intestine and more severe abdominal pain (Figure 153).

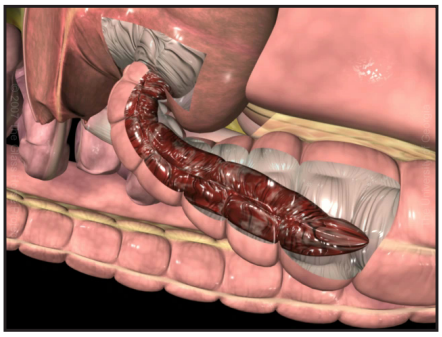

Figure 153. Strangulation of the cecum in the right ventral colon

Diagnosis of cecocolic intussusception can be difficult, although rectal examination may reveal a **mass in the right caudal region of the abdomen** in about half of affected horses. **Transabdominal ultrasonography** can be used to identify bowel within bowel, a characteristic finding with intussusceptions. In many cases, the diagnosis is made during exploratory celiotomy rather than preoperatively. Surgical intervention is required but is not always successful due to peritonitis, rupture of the cecum, or an inability to reduce the intussusception. The approach may require partial typhlectomy either after manual reduction of the intussusception or through a colotomy incision. The prognosis for survival is fair, but it can be improved if appropriate steps are taken to minimize contamination during surgery and supportive care is intensive.

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Ascending or Large Colon

Pelvic Flexure Impaction (Obstruction)

Normally, the ingesta is mixed and digested in the ventral colons. Nutrients generated by the digestion process are absorbed, water is gradually reabsorbed, and the undigested material moves into the dorsal colons. An impaction can occur when dry or inadequately digested feed fails to move through the **pelvic flexure**, the region connecting the large left ventral colon with the smaller left dorsal colon. The reduction in diameter of the colon at the pelvic flexure is dramatic, making this a natural site for development of an obstruction (Figure 154). Because the impacted feed material cannot pass through the pelvic flexure, additional ingesta fills the entire **left ventral colon** (Figure 155). As this occurs, the impacted colon elongates and the pelvic flexure region moves into the pelvic inlet (Figure 156). Because one of the functions of the large colon is to reabsorb water from the intestinal contents, the ingesta in the pelvic flexure and left ventral colon becomes even drier, and the colonic wall becomes tightly adhered to the ingesta (Figure 157). The horse feels pain as a result of the stretching of the colonic wall and contraction of the colonic muscles against the impacted material.

Most affected horses intermittently exhibit signs consistent with **mild abdominal pain** and are not toxemic. The heart rate usually is only slightly increased. Intestinal sounds usually can be heard, presumably as the intestinal muscles work against the obstruction, and may be associated with signs of

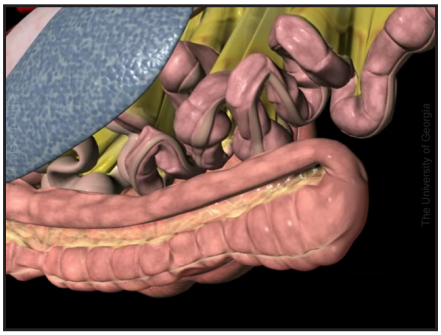

Figure 154. Impaction of ingesta at the pelvic flexure

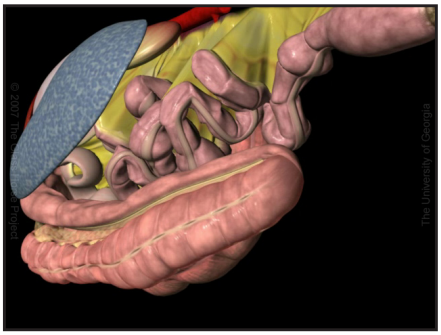

Figure 155. Filling of the left ventral colon with ingesta

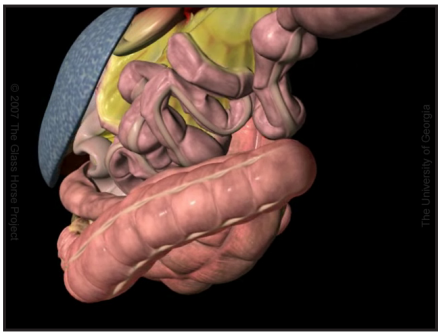

Figure 156. Movement of the pelvic flexure into the pelvic inlet

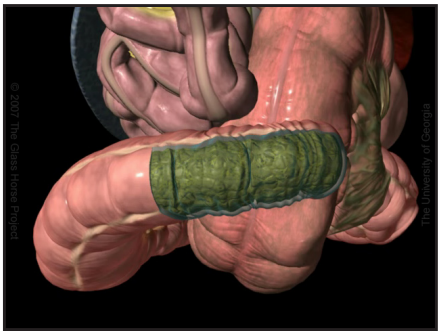

Figure 157. Reabsorption of water from the ingesta

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pain. Treatment involves oral and intravenous administration of fluids, mild analgesics, and laxatives. Feed is withheld until the impaction has resolved. If the impaction is not resolved after several days of medical management, surgical intervention may be necessary. However, it is the rare case that requires surgical treatment, and the prognosis for survival is excellent.

Sand Impaction (Obstruction)

Sand impactions are commonly encountered in horses fed on sandy soils, with affected horses developing clinical signs reflecting one of two scenarios. In one scenario, the sand accumulates in the **right dorsal colon** and eventually obstructs the junction of the right dorsal and transverse colons (Figure 158). This results in **severe distention** of the colon proximal to that junction and signs of severe abdominal pain (Figure 159). In the other scenario, sand accumulates in the dependent portions of the colon. The weight of the sand causes the **sacculations to flatten** (Figure 160), and the **mucosa thickens** as a response to chronic irritation (Figure 161); both of these features can be recognized by ultrasonography. Horses in which the sand accumulates in the ventral colon show signs consistent with mild abdominal pain. These horses may stretch out or spend time lying down, presumably to reduce the effect of the weight of the sand on the colonic mesentery. In some horses the sand causes diarrhea, presumably by irritat-

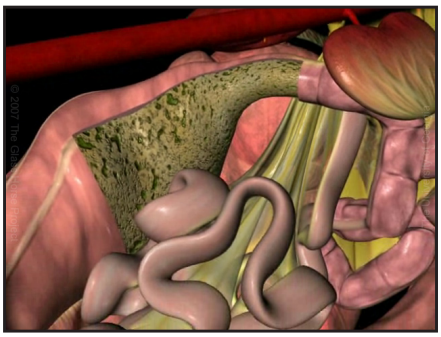

Figure 158. Obstruction of the right dorsal/transverse colon junction

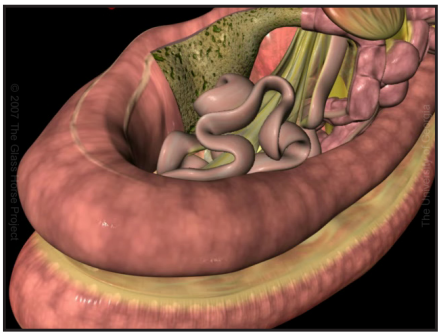

Figure 159. Distention of the ascending colon proximal to the obstruction

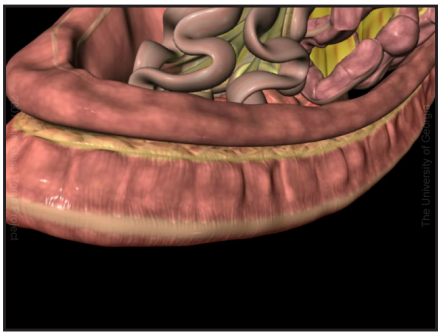

Figure 160. The weight of sand flattens the sacculations in the ventral colon.

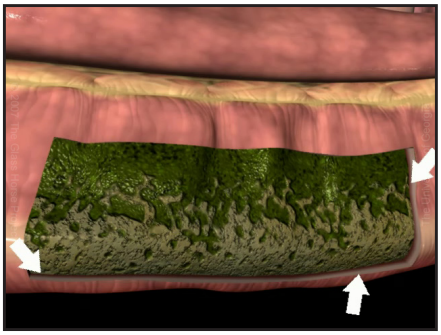

Figure 161. Thickening of the colonic mucosa

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ing the mucosa. Sand may be palpable in the feces and will be evident if the feces are mixed with water and allowed to settle.

If the degree of pain is mild, therapy is directed towards removing the sand and ingesta from the colon by repeated administration of psyllium methylcellulose, oral and intravenous fluids, and analgesics. If pain is severe and unrelenting, surgery is necessary to remove the sand from the right dorsal and transverse colons. The prognosis for most horses with sand colic is good, although complications may arise during surgery as it is extremely difficult to remove the sand from the transverse colon region.

Large Colon Volvulus (Strangulation Obstruction)

Although often referred to as a **large colon torsion**, this condition is actually a volvulus because the twisting involves the mesentery between the ventral and dorsal colons. This condition occurs most often in **broodmares** around 8 years old and classically occurs approximately **3 months after foaling**. Twisting of the colon occurs at the level of the cecocolic mesentery, with the right ventral colon rotating medially and dorsally (Figure 162). When viewed from the caudal aspect of the horse, the twist occurs in a clockwise direction. The severity of the twist may vary from 270 degrees to 720 degrees (Figure 163), with the end result being obstruction of the lumen of the colon and ischemia of the majority of the ventral and dorsal colons. Occlusion of the lumen of the colon results in **rapid accumulation of gas**, distention (Figure 164), and severe pain, and **ischemia** results in the development of endotoxemia. The onset of abdominal pain associated with this condition is sudden and may be unresponsive to analgesics. The heart rate increases, and there is rapid deterioration of peripheral perfusion. Marked abdominal distention is usually present.

Treatment of affected horses requires immediate surgery to correct the volvulus and, if necessary, remove affected bowel. Because there is a recurrence rate of about 15% in broodmares, many surgeons elect to perform a colopexy procedure or to remove half of the large colon in broodmares in which the condition has occurred a second time. Although the condition carries a poor prognosis in many regions of the country, survival rates are excellent in practices near large breeding farms. This difference in prognosis reflects differences in the time required to transport the mare to a clinic where surgery can be performed.

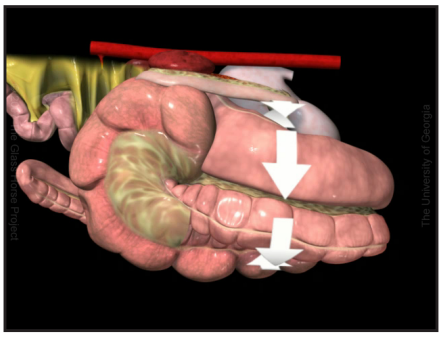

Figure 162. The direction of colonic volvulus

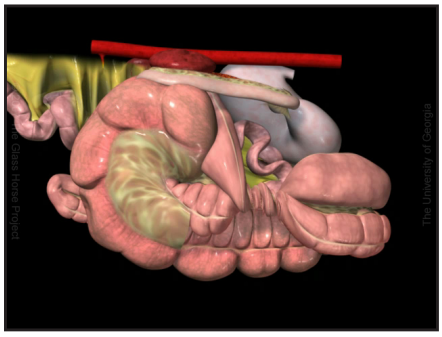

Figure 163. Obstruction and strangulation of the large colon

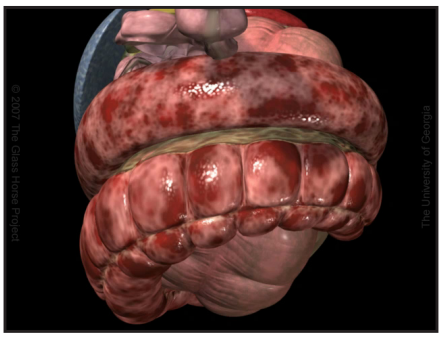

Figure 164. Distention of the strangulated colon

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Large Colon Enterolithiasis (Obstruction)

Enteroliths are concretions of **magnesium ammonium phosphate** that develop in the large colon around a nidus, such as a piece of stone or metal. In most cases, large individual enteroliths that exceed the diameter of the **transverse colon** cause acute obstruction of the ascending colon (Figure 165). The colon proximal to the obstruction distends with gas (Figure 166), causing the horse to become painful. Although enteroliths may be quite large, most often they are not palpable on rectal examination as they lodge in the transverse colon, cranial to the cranial mesenteric artery. In some horses more than one enterolith may develop in the large colon. When this happens, the enteroliths rub together, causing their sides to become flattened; as a result, the stones become **polyhedral** in shape. In most cases, one of these stones lodges in the proximal part of the descending colon, causing obstruction of the colon and abdominal pain (Figure 167). In the United States, enterolithiasis occurs most commonly in the Southwest, California, Florida, and Indiana, with **Arabian and Arabian-cross horses** being over-represented among affected horses. Most affected horses are at least **10 years old**, and there is a suspected association with ingestion of **alfalfa hay**, presumably due to its high concentration of magnesium.

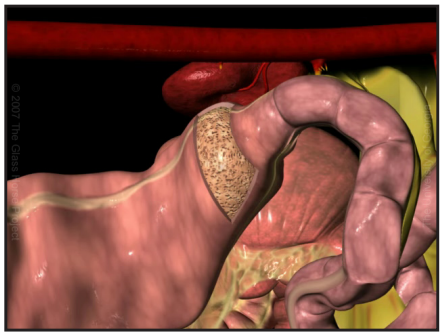

Figure 165. Obstruction of the ascending colon by a single enterolith

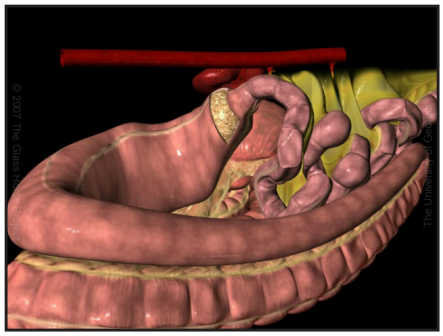

Figure 166. Distention of the ascending colon proximal to the obstruction

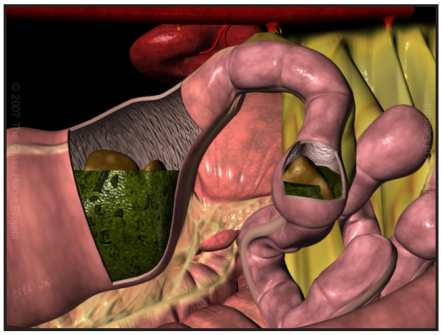

Figure 167. Obstruction of the descending colon by a polyhedral stone

Many horses suffering from large colon enterolithiasis have a history of recurring bouts of acute pain, and in some horses the enterolith can be identified with either radiography or ultrasonography. Treatment requires surgical intervention to decompress colon and cecum and then to remove the enterolith(s). If an enterolith has a flat side or a polyhedral shape, one should always check for additional enteroliths. The prognosis for survival is excellent in areas where enterolithiasis is common.

Left Dorsal Displacement (Obstruction)

Left dorsal displacement occurs when the **large colon** becomes lodged between the **spleen** and **left kidney**. In this condition, the left colon moves dorsally and becomes entrapped between the spleen, the renosplenic ligament, and the left kidney (Figure 168). As the colon becomes entrapped, it rotates on its axis such that the ventral colon lies more dorsally and the dorsal colon lies more ventrally (Figure 169). Due to its large size, the colon may become hooked over the dorsal edge of the spleen.

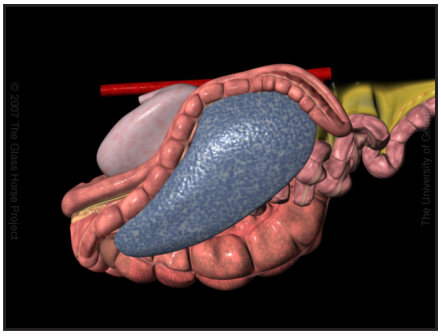

Figure 168. Dorsal displacement of the large colon

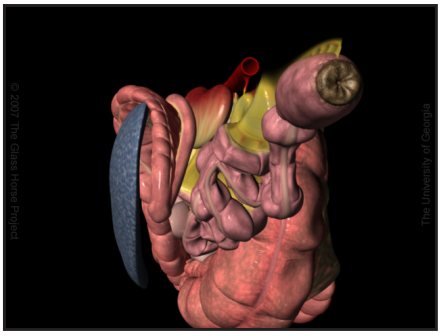

Figure 169. Entrapment of the colon by the renosplenic ligament

If the displacement occurs such that the weight of the pelvic flexure is borne by the renosplenic ligament, the **spleen rotates** away from the left body wall (Figure 170). The weight of the colon in this area also may impair blood flow through the splenic vein at the hilus, resulting in engorgement of the spleen with blood (Figure 171). This **engorgement** of the spleen accounts for the fact that **splenic blood** may be retrieved via **abdominocentesis** in some

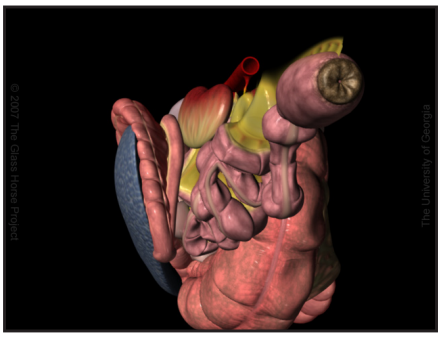

Figure 170. Rotation of the spleen away from the left body wall

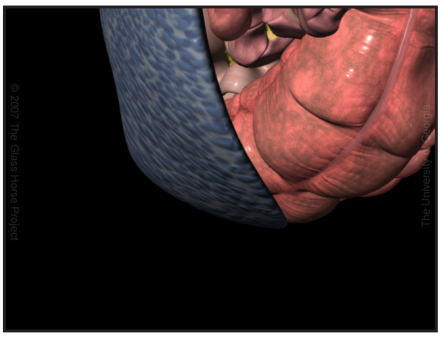

Figure 171. Engorgement of the spleen

horses. Because the flow of ingesta in the colon is impeded, an impaction of the left dorsal colon may develop secondarily as water is reabsorbed.

Affected horses typically exhibit mild to moderate abdominal pain and may have a prolonged course of intermittent painful episodes. If, however, the colon distends with gas, the severity of the pain will increase. The diagnosis is made by a combination of **rectal examination** and **ultrasonography**. On rectal examination, longitudinal bands of the left ventral colon may be palpated

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running dorsocranially to the left kidney. It may not be possible to identify the kidney or the most dorsal aspect of the spleen using ultrasonography, and ultrasonography should not be used alone to diagnose this condition.

There are **four possible treatments** for left dorsal displacement of the large colon: feed restriction; administration of phenylephrine to cause contraction of the spleen; short-term anesthesia and rolling; and finally, surgical intervention. Surgery is generally reserved for those horses that fail to respond to other treatments or that are acutely painful. The prognosis for survival is excellent.

Right Dorsal Displacement (Obstruction)

Right dorsal colon displacement involves **displacement of the colon between the cecum and right body wall**. This condition is preceded by development of a pelvic flexure impaction in which the pelvic flexure is displaced cranially in the abdomen (Figure 172). Gas distention of the sternal and diaphragmatic flexures then causes the colon to migrate caudally along the ventral abdominal wall (Figure 173). In most cases, the

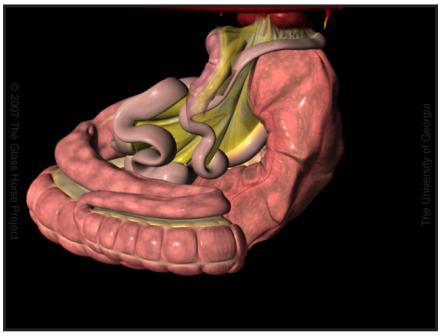

Figure 172. Retroflexion of the pelvic flexure

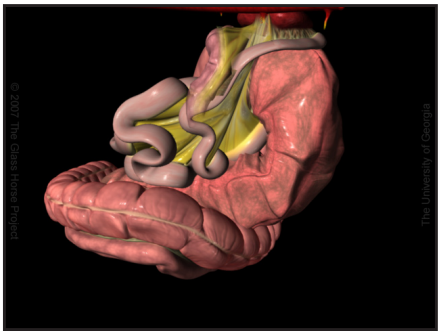

Figure 173. Caudal migration of the large colon ventral to the cecum

colons also twist on their long axes at the level of the cecocolic ligament, rendering the affected colon edematous but not ischemic. The colon comes to its final position caudal to the cecum (Figure 174). Approximately 50% of horses with this displacement have increased serum concentrations of gamma glutamyl transferase, a finding that has been associated with stretching of the mesoduodenum and compression of the bile duct in the hepatoduodenal ligament.

Affected horses exhibit variable degrees of pain depending on the degree of distention of the colon. In some horses the abdomen will be distended and tight due to the gas in the obstructed colon; these horses tend to show signs of moderate abdominal pain. In other horses, there is minimal

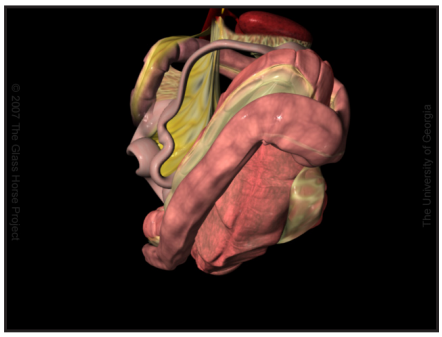

Figure 174. Right displacement of the ascending colon from the rectal point of view

abdominal distention and evidence of mild pain. Rectal examination may reveal teniae of the **colon running transversely** across the pelvic inlet, and the examiner may not be able to identify the cecum or pelvic flexure (Figure 175). In horses with mild abdominal pain, the condition often can be managed medically. The decision to perform surgery usually is based on the continued presence of pain and abnormal rectal findings. The prognosis is very good, with most clinics reporting greater than 75% survival rates.

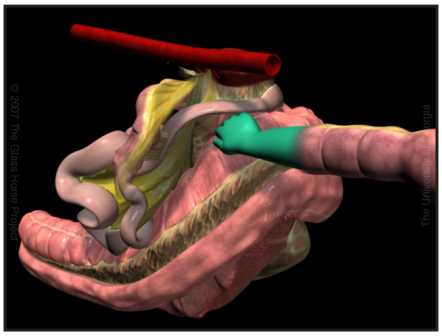

Figure 175. Identification of the colon transversely across the pelvic inlet

Colitis (Colitis)

The term colitis refers to **inflammation of the wall of the colon** (Figure 176). The principal clinical findings in horses with colitis are diarrhea, fever and signs of endotoxemia (increased heart rate, prolonged capillary refill time, discolored mucous membranes). There are numerous causes of colitis in horses, ranging from bacteria to sand to adverse responses to specific drugs. Very often, it is difficult, if not impossible, to identify the specific cause in individual horses. Some horses with colitis are acutely painful, which makes the diagnosis even more difficult as treatment of colitis is considerably different from treatment of abdominal pain due to a colonic displacement or volvulus. The pain associated with colitis is due to a gas-distended or impacted large colon or cecum, hypermotility or ileus, and edema, inflammation, or infarction of the intestinal wall (Figure 177). If the diarrhea is severe, signs of hypovolemic shock will ensue.

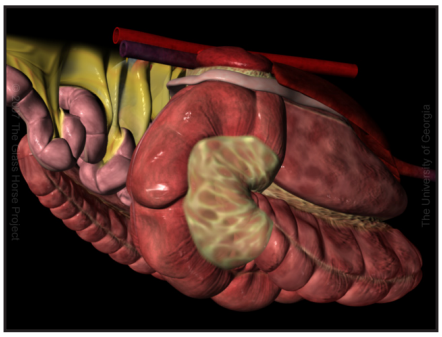

Figure 176. Inflammation of the wall of the colon

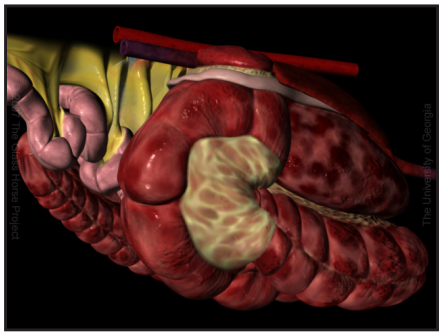

Figure 177. Inflammation, edema, and infarction of the colonic wall

The most common clinicopathologic findings in horses with acute colitis are **neutropenia with a left shift**, the presence of toxic neutrophils, metabolic acidosis, azotemia, hypoproteinemia, hyponatremia, hypochloremia, and hypokalemia. Transabdominal ultrasound may reveal a thickened colon or cecal wall, and analysis of peritoneal fluid typically reveals a normal nucleated cell count and normal to mildly increased protein concentration. **Peritonitis** can develop if edema or inflammation of the intestinal wall is marked, allowing the movement of enteric bacteria into the peritoneal cavity. The most commonly encountered causes of acute colitis in horses include salmonellosis, Potomac Horse Fever, nonsteroidal anti-inflammatory drug toxicity, cyathostomiasis, sand ingestion, and infectious with clostridial organisms.

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Nonstrangulating Infarction

The term **nonstrangulating infarction** refers to a condition in which blood flow is reduced to a portion of intestine in the absence of a concurrent displacement or incarceration. The end result is infarction of the affected intestine (Figure 178), which may be the cecum, ascending colon, or small intestine. This condition was encountered far more commonly in years past when parasite control was inadequate.

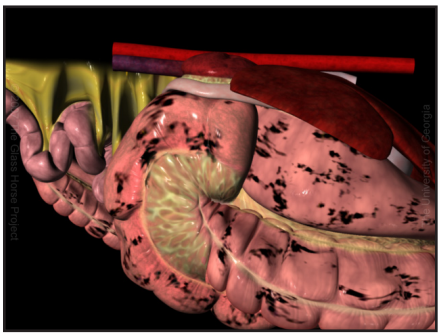

Figure 178. Widely dispersed lesions typical of nonstrangulating infarction.

The cause of nonstrangulating infarction may be **thromboembolism** or a **reduction in local blood flow** in intestinal wall causing insufficient delivery of blood to the intestine. Although the condition is presumed to occur secondary to parasitism, it also may be seen in horses with good parasite control and in those who have undergone gastrointestinal surgery for conditions such as **large colon volvulus**.

There are two clinical scenarios associated with this disease. Some affected horses may have chronic intermittent episodes of mild to moderate abdominal pain without any evidence of intestinal obstruction. These horses periodically are depressed and there may be evidence of deterioration of the systemic circulation (i.e., altered mucous membrane color and prolonged capillary refill time). In other horses, there may be complete infarction of the intestine with or without evidence of a thrombus. These horses may become very painful and have a distended colon that can be palpated during the rectal examination. In these cases, a paracentesis may yield fluid with very **high leukocyte counts** (greater than 200,000 cells/µl) and containing numerous red blood cells.

Affected horses are treated symptomatically with analgesics and intravenous fluid replacement, and, if necessary, larvicidal therapy using appropriate anthelmintic drugs. Although heparin and/or aspirin have been suggested as potential components of the treatment plan, the number of cases seen is so small that it is difficult to determine whether or not these treatments are efficacious. Overall, the prognosis for survival is **guarded to poor**, depending on the amount of intestine involved.

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Descending or Small Colon

Enterolithiasis (Obstruction)

Enteroliths are concretions of magnesium ammonium phosphate crystals that develop in the large colon around a nidus, such as a piece of stone or metal. Small **polyhedral-shaped enteroliths** formed in the large colon may pass into the small colon where they obstruct the lumen, causing **gas distention** proximal to the obstruction (Figure 179). Local ischemia of the small colon wall also may occur due to excessive pressure of the enterolith against the wall.

Many horses with this condition have a history of recurring acute bouts of pain. Depending on the size of the horse, some enteroliths can be identified on radiographs of the abdomen. Treatment requires surgical intervention to remove the obstructing enterolith. If an enterolith has a flat side or a polyhedral shape, one should always check for additional enteroliths. The prognosis is excellent in areas where enterolithiasis is common.

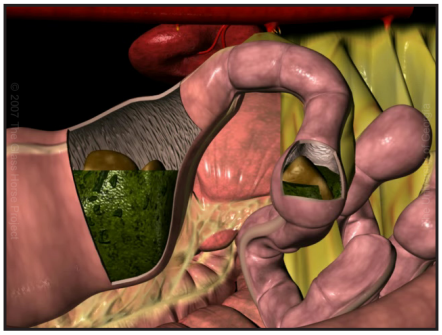

Figure 179. Obstruction of the descending colon by a polyhedral stone

Impaction (Obstruction)

Impaction is the most commonly encountered condition affecting the small colon, and it occurs commonly in **American Miniature Horses** and **Shetland ponies**. As the impaction develops, the fecal balls and sacculations that characterize the descending colon are replaced by a long tube of dehydrated ingesta (Figure 180). Due to its weight, the impacted descending colon sinks to the **caudoventral aspect of the abdomen** (Figure 181), and the descending colon proximal to the impaction distends with gas (Figure 182).

Generally, the degree of abdominal pain is mild to moderate, but it may be severe if the obstruction is complete. Tympany of the colon and cecum occurs secondarily to the obstruction, and ileus results. It may be possible to feel the impaction during the rectal examination, or one may only feel distention of the colon proximal to the obstruction. Impactions

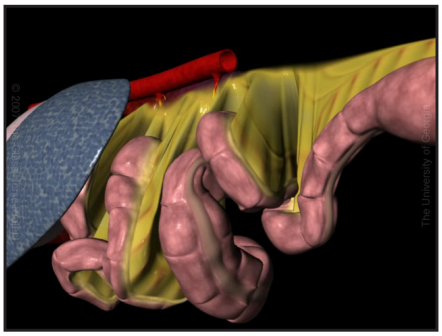

Figure 180. Obstruction of the descending colon by dry ingesta

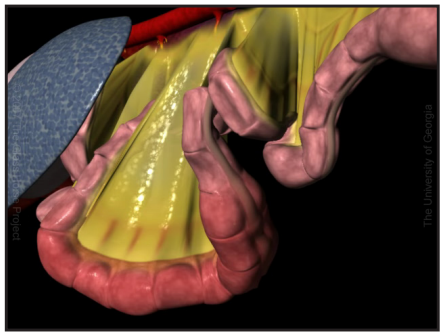

Figure 181. Ventral displacement of the impacted descending colon

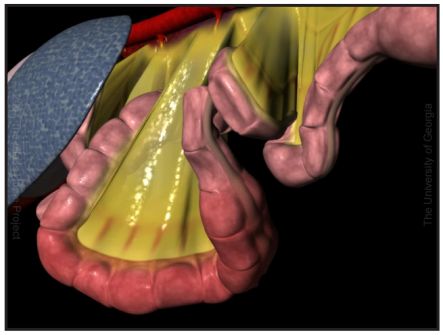

Figure 182. Distention of the descending colon proximal to the impaction

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occur far more often in the fall and winter months, and horses with small colon impactions also may have diarrhea. Approximately 50% of horses with small colon impactions that undergo surgery have **positive fecal cultures for Salmonella**, requiring these horses to be isolated from other horses in the clinic. While medical treatment may be successful in many cases, surgical intervention often is needed due to the severity of pain and gas distention. The prognosis for survival is very good.

Liver

Cholelithiasis (Obstruction)

Cholelithiasis, the formation of **biliary calculi**, causes fever, icterus, and intermittent episodes of mild abdominal pain. Most affected horses are **more than 3 years of age**. Although the condition may be due to a single large cholelith (Figure 183), it more commonly results when multiple choleliths obstruct bile ducts within the liver (Figure 184). Most often, cholelithiasis is diagnosed by ultrasonography, with dilated ducts and hepatoliths being the most common findings. There is evidence of ascending bacterial infections in many cases, and serum concentrations of gamma glutamyl transferase are extremely high. Treatment may involve a combination of medical and surgical interventions, and the prognosis is highly dependent on the duration of the condition before it is recognized.

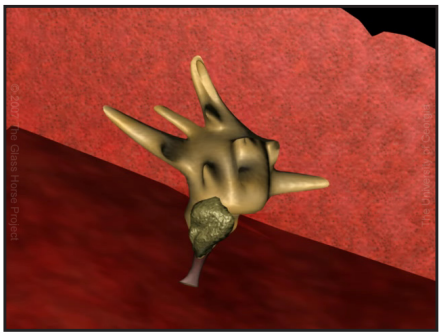

Figure 183. Obstruction of the bile duct by a single large cholelith

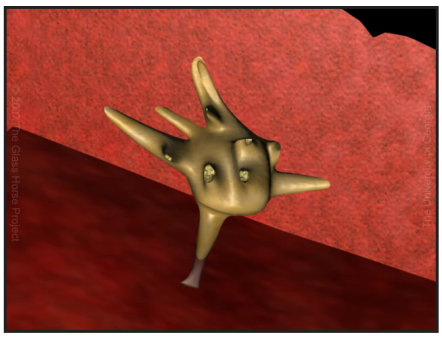

Figure 184. Obstruction of bile ducts in the liver by multiple small choleliths

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